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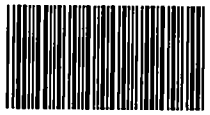
Chino Mines Company
Administrative Order on Consent
Lampbright Investigation Unit
Human Health Risk Assessment
Revision 1

Prepared by:
Neptune and Company, Inc.

For:
New Mexico Environment Department



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Executive Summary

A baseline human health risk assessment (HHRA) has been conducted by Neptune and Company, Inc. (Neptune) for the Chino Mines Company (Chino) Lampbright Investigation Unit (LIU; near Hanover, New Mexico) to evaluate the potential for adverse human health effects associated with historical mining operations.

The HHRA provides the best information possible to make informed and expedient risk-based decisions regarding the LIU. The HHRA will assist the involved parties (New Mexico Environment Department [NMED], Chino, and the public) in making decisions regarding remediation and risk management at the site, in accordance with the Administrative Order on Consent (AOC) entered on December 23, 1994. A site-wide ecological risk assessment has been completed by another contractor.

The HHRA described in this report follows a two-tiered approach. The screening-level Tier I assessment assesses maximum detected concentrations of constituents of interest (COIs) in exposure equations that include conservative (i.e., biased toward protection of human health) exposure and chemical toxicity assumptions. This Tier I assessment identifies constituents of potential concern (COPCs) carried forward to the Tier II HHRA, which includes refined assumptions.

The COIs/COPCs for which risk is assessed in the LIU HHRA are limited to metal compounds, per prior agreement between NMED and Chino [Chino, 2010]. The HHRA is based upon environmental data collected during the RI and previous investigations. The Remedial Investigation (RI) Report for the LIU [Arcadis, 2012] contains a substantial amount of historical and background information. This information will not be repeated here except as necessary for context.

Briefly, the LIU is located in the northeast corner of the overall AOC Investigation Area, south of State Highway 152 and east of the operational Santa Rita Open Pit. The LIU includes the area surrounding the present Lampbright Leach Stockpile that may be affected by historic operations. Specifically, it includes "Tributary 1" downgradient of Dam 8 (which forms Reservoir 8, a pregnant leach solution [PLS] collection area), the North Cut Diversion Area, and "Tributary 2" plus other downgradient areas. Surrounding upland areas are also included in the LIU. There are a variety of potential sources and release mechanisms of COI contamination associated with the LIU that were investigated in the RI. Primary sources of environmental releases include the Lampbright Stockpile Area (LSA), the solution extraction/electrowinning (SX/EW) plant; and the PLS system, including pipelines, collection tanks, and reservoirs. The sources of interest for the HHRA (under the AOC) are the LSA and fugitive leach solution. Primary release mechanisms include fugitive dust, spray (from historic leach water emitters on the LSA), rainwater seepage, spills, and storm water events. Transport mechanisms include infiltration and percolation in the LSA, overland flow of contaminated water, resuspension via stormwater, and accidental spills of PLS and process water. Secondary sources include upland soil impacted by fugitive dust, tributary sediment impacted by dust or runoff, and biotic (i.e., plant and animal) uptake.

A conceptual site model (CSM) was developed that functions as a mechanism for integrating COI/COPC sources, release mechanisms, secondary sources, transport mechanisms, intermediate exposure media, final exposure media, exposure routes, and receptors (i.e., persons who may be exposed at the site). The most likely exposure scenarios, based upon current land use, previous HHRAs, a tour of the overall site, and discussions with Chino and NMED, include:

A. Present and future commercial ranching;
B. Present trespassing on Chino property;
C. Future residential development;
D. Future recreation (e.g., hiking); and,
E. Future construction work.

Receptors may be exposed to constituents via ingestion of dust/soil and water, inhalation of dust, and dermal (skin) absorption.

Based upon the Tier I screening of COIs, COPCs evaluated in the Tier II HHRA include aluminum, arsenic, hexavalent chromium (CrVI), cobalt, and manganese. Comparisons of LIU site concentrations of these COPCs with concentrations at the LIU reference area plus an area used in a previous HHRA reveal little statistical differences between impacted areas and relatively non-impacted areas.

The Tier II HHRA found that the only potential issue from a human health perspective may be nervous system effects related to manganese concentrations in soils in a future construction scenario. However, this is likely due to highly conservative assumptions regarding the quantity of dust generated by vehicle traffic on unpaved roads used in the exposure assessment. Manganese site concentrations do not appear to be elevated above the LIU reference data, and marginally elevated above the STSIU/ERA reference data. Therefore, it is not informative to estimate preliminary remediation goals at this time.

If the estimated risks or levels of uncertainty are unacceptable to the involved parties, then it may be informative to conduct a more detailed probabilistic (i.e., Monte Carlo simulation) assessment to identify the degree of conservatism associated with the Tier II assessment and to identify important sources of uncertainty. This may also include further collection or analysis of site data.

1 Introduction

1.1 Regulatory Context

The LIU is one of six original IUs within the AOC Investigation Area (see [Arcadis, 2012], Figure 1-1), which are all subject to distinct HHRAs. The RI process is conducted under the AOC between Chino and NMED. The AOC (effective December 23, 1994) addresses effects of historical operations from Chino's copper mining and processing facilities within the AOC Investigation Area.

In addition to the AOC, New Mexico Water Quality Control Commission regulations require development of a discharge plan for discharge of effluent or leachate to prevent adverse impact upon groundwater resources [Chino, 1995]. Some of the activities to be addressed under the AOC for LIU are being addressed under discharge permit (DP)-related programs (i.e., Sitewide Abatement and the DP-376 Corrective Action). Based upon prior agreement between Chino and NMED, the LIU HHRA focuses upon issues outside of those covered under DP-related programs.

In addition to the discharge plan, Chino is also regulated under Clean Water Act regulations for stormwater discharges via EPA's Multi-Sector General Permit program. However, Chino collects and manages all stormwater that contacts stockpiles and tailing impoundments on-site. Thus, these facilities are effectively zero-discharge for surface water. Only particular areas at Chino are authorized to discharge storm water, and these areas are limited to plant operation and maintenance areas, access roads and material storage areas located outside of Chino's zero-discharge area.

1.2 Purpose and Objectives

The basic approach taken in the HHRA is that defined by the United States Environmental Protection Agency's (EPA's) Risk Assessment Guidance for Superfund, Human Health Evaluation Manual, Part A [EPA, 1989]. The components of this approach involve data collection and evaluation, exposure assessment, toxicity assessment, and risk characterization.

The HHRA incorporates a two-tiered approach. The Tier I assessment involves screening of COIs using maximum detected concentrations and conservative assumptions. A type of screening assessment was performed in the RI (i.e., comparison with EPA Regional Screening Levels, or RSLs [EPA, 2012c], but is expanded for the purpose of the HHRA. The reason is that RSLs are not fully informative for screening at the LIU. EPA RSL guidance states the following (numbers added for clarity):

> *As with any risk based screening table or tool, the potential exists for misapplication. In most cases, this results from not understanding the intended use of the SLs or PRGs. In order to prevent misuse of the SLs, the following should be avoided:*
>
> 1. *Applying SLs to a site without adequately developing a conceptual site model that identifies relevant exposure pathways and exposure scenarios.*

2. *Not considering the effects from the presence of multiple contaminants, where appropriate.*
3. *Use of the SLs as cleanup levels without adequate consideration of the other NCP remedy selection criteria on CERCLA sites.*
4. *Use of SL as cleanup levels without verifying numbers with a toxicologist or regional risk assessor.*
5. *Use of outdated SLs when tables have been superseded by more recent values.*
6. *Not considering the effects of additivity when screening multiple chemicals.*
7. *Applying inappropriate target risks or changing a cancer target risk without considering its effect on noncancer, or vice versa.*
8. *Not performing additional screening for pathways not included in these SLs (e.g,. vapor intrusion, fish consumption).*
9. *Adjusting SLs upward by factors of 10 or 100 without consulting a toxicologist or regional risk assessor.*

Similar language is found in recent NMED soil screening level guidance [NMED, 2012]. It is necessary that the most appropriate and scientific means be applied to the important issue of screening potential human health risks at the LIU. Tier 1 of the HHRA therefore follows an appropriate site-specific screening mechanism to identify COPCs.

The Tier II assessment focuses upon the specific COPCs that are the primary sources of potentially unacceptable human health risks (based upon the Tier I screening), and involves refined COPC concentrations and receptor (i.e., a type of person exposed at the site) and exposure-pathway specific calculations. The Tier II assessment informs decisions as to whether unacceptable human health risks associated with the LIU under current and future land uses may exist, as well as any risk management or remediation decisions.

The Tier I and Tier II components of the present HHRA are 'deterministic'; i.e., they estimate single-point values for risk in different scenarios, as opposed to a 'probabilistic' analysis, which estimates distributions or ranges of values via incorporation of variability and uncertainty associated with assumptions. If potentially unacceptable risks are found in the initial Tier II HHRA, then it may be informative to conduct a more detailed probabilistic (i.e., Monte Carlo simulation) assessment to identify the degree of conservatism associated with the Tier II assessment and to identify important sources of uncertainty. This may also include further collection or analysis of site data.

The HHRA focuses upon providing the best information possible to make informed and expedient decisions regarding the LIU. However, compared to other Chino IUs the LIU is 'simpler' in terms of geomorphology (i.e., characteristics of the land) and has no current residents, thus NMED does not consider it necessary for the LIU HHRA to be as complex and extensive as previous Chino risk assessments. Regardless, this HHRA is conducted according to regulatory guidance in a scientifically defensible manner.

The following sections describe the essential components of a HHRA:

- Conceptual site model (CSM)
- Data evaluation for COIs
- Exposure assessment
- Toxicity assessment
- Risk characterization
- Uncertainty assessment
- Conclusions

2 Conceptual Site Model

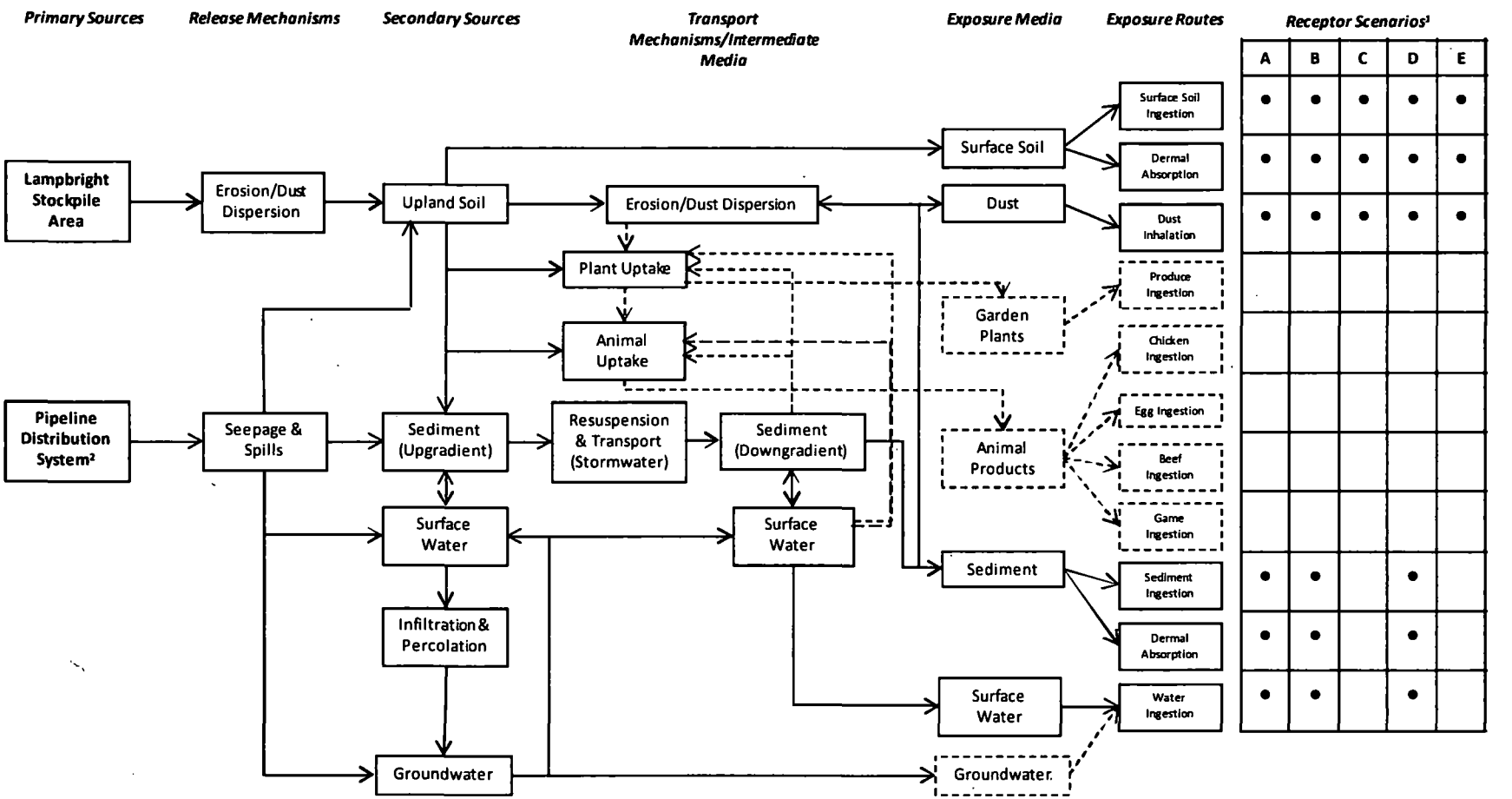
2.1 Overview

The CSM functions as a mechanism for integrating COI sources, release mechanisms, secondary sources, transport mechanisms, intermediate exposure media, final exposure media, exposure routes, and receptors. According to EPA [EPA, 1989]:

> *An exposure pathway generally consists of four elements: (1) a source and mechanism of chemical release, (2) a retention or transport medium (or media in cases involving media transfer of chemicals), (3) a point of potential human contact with the contaminated medium (referred to as the exposure point), and (4) an exposure route (e.g., ingestion) at the contact point. A medium contaminated as a result of a past release can be a contaminant source for other media (e.g., soil contaminated from a previous spill could be a contaminant source for ground water or surface water). In some cases, the source itself (i.e., a tank, contaminated soil) is the exposure point, without a release to any other medium. In these latter cases, an exposure pathway consists of (1) a source, (2) an exposure point, and (3) an exposure route.*

The CSM forms the overall framework for the HHRA. The major components of the CSM are a physical model of the IU and an exposure model. The RI [Arcadis, 2012] contains figures indicating the location and scale of the site (e.g., Figure 1-1). Figure 1 below is a graphical representation of the LIU CSM. Note that there are a number of media types and exposure routes that were qualitatively considered but not quantitatively assessed in the HHRA, as explained below.

Figure 1: Conceptual Site Model for the LIU Human Health Risk Assessment



Notes:

All transport mechanisms, exposure media, and exposure routes that are complete are indicated by solid lines and boxes; those that are incomplete or otherwise not considered are indicated by dashed lines and boxes. See text for explanation.

1: Receptor scenarios defined in Table 1. A: commercial ranching; B: trespassing; C: residence; D: recreation; E: construction. Dots indicate considered pathways.

2: "Pipeline distribution system" refers only to historical use of mine water used as leach solution, and not to current operations.

2.2 Physical System Model

2.2.1 Description

The purpose of developing a model of the physical system is to define the key processes and features in the environment that are believed to control COI distribution within the LIU. This includes all the processes in the CSM leading to exposure point concentrations (EPCs) in final exposure media (i.e., COI sources, release mechanisms, secondary sources, transport mechanisms, and intermediate exposure media).

2.2.2 Types of Contamination

Contamination addressed in the HHRA includes analyzed metal compounds associated with historical mining operations and releases. All of these metals are also naturally-occurring. Although other types of chemicals were and are employed in mining operations (e.g., raffinate), these chemicals are typically captured to the extent possible for re-use, and any water releases are covered under the DP. Thus, NMED and Chino have agreed to focus upon metals for the RI and the HHRA. The list of COIs includes the following:

- Aluminum
- Arsenic
- Barium
- Beryllium
- Boron
- Cadmium
- Chromium (see text below)
- Cobalt
- Copper
- Iron
- Lead
- Manganese
- Mercury
- Molybdenum
- Nickel
- Selenium
- Silver
- Thallium
- Vanadium
- Zinc

The RI evaluated total chromium; i.e., it did not differentiate different valence states or chemical forms. As the most common forms of chromium (trivalent: CrIII; and hexavalent: CrVI) have very different toxicity, it is necessary to assume that total chromium in media other-than water is comprised of some ratio of these forms. Briefly, EPA [EPA, 2012a] assumed for the derivation of the "inhalation unit risk" value that a ratio of CrIII to CrVI of 6:1 in air existed in the workplace at the location associated with

the critical dose-response study. This ratio has been used in previous HHRAs at the site (as well as numerous other HHRAs in the United States of America [US]), and will be assumed here in lieu of site-specific information (not available at this time). For surface water, it is assumed that all chromium is CrVI, as CrIII is marginally soluble.

The following analytes are not explicitly evaluated here. Antimony was previously analyzed in surface water and plant tissue; however, it was not detected. Magnesium was also analyzed in soils and sediments; but it has no published toxicity values. Potassium, sodium, and sulfate were analyzed, but are generally not considered to be toxic in environmental concentrations. Lead is toxic, but is addressed differently from other COIs (i.e., lead has distinct screening levels), as discussed further below.

2.2.3 Lampbright Investigation Unit Description

The LIU is located in the northeast corner of the overall AOC Investigation Area, south of State Highway 152 and east of the operational Santa Rita Open Pit (see Figures 1-1 and 2-1 in [Arcadis, 2012]). The LIU includes the area surrounding the present Lampbright Leach Stockpile that may be affected by historic operations. Specifically, it includes "Tributary 1" downgradient of Dam 8 (which forms Reservoir 8, a pregnant leach solution [PLS] collection area), the North Cut Diversion Area, and "Tributary 2" plus other downgradient areas. Note that these drainages are generally ephemeral, with flow occurring only during storm and spring runoff events (however, pools may exist for extended periods). Surrounding upland areas are also included in the LIU.

2.2.4 Contamination Sources, Releases, and Transport

There are a variety of potential sources and release mechanisms of COI contamination associated with the LIU that were investigated in the RI. These are roughly categorized as primary and secondary.

Primary sources of environmental releases include the Lampbright Stockpile Area (LSA), the solution extraction/electrowinning (SX/EW) plant; and the PLS system, including pipelines, collection tanks, and reservoirs. The sources of interest for the HHRA (under the AOC) are the LSA and fugitive leach solution. Primary release mechanisms include fugitive dust, spray (from raffinate emitters on the LSA), rainwater seepage, spills, and storm water events. Transport mechanisms include infiltration and percolation in the LSA, overland flow of contaminated water, resuspension via stormwater, and accidental spills of PLS and process water. Secondary sources include upland soil impacted by fugitive dust, tributary sediment impacted by dust or runoff, and biotic (i.e., plant and animal) uptake.

In the HWCIU and STSIU HHRAs [Neptune, 2008;Gradient, 2008], food pathways (e.g., home-grown produce, chickens, beef, etc.) were evaluated. In the case of the LIU, NMED has determined that modeling potential exposures related to home-raised foods and game would provide limited information for risk management in the LIU. NMED has decided not to pursue the foodstuff pathways in the LIU HHRA because of the low likelihood that future residents would engage in extensive agricultural activities or gather extensive site-related game, and because cultivation of produce will likely require appreciable amendments for productive garden soil due to the poor quality of existing soil.

Potential groundwater impacts are not evaluated in the HHRA, as these are addressed under a different regulatory construct; i.e., Discharge Plan (DP) 376/Corrective Action and Site Wide Abatement. The RI report [Arcadis, 2012] contains details. Any exceedences of groundwater criteria would be addressed under that regulatory construct. It is assumed here that any potential residential groundwater use in upland areas would not be likely to be impacted by site activities; but regardless any such impacts would be addressed under the DP. However, the implications of this assumption will be discussed in the Uncertainty Assessment section.

Factors affecting the extent of present contamination include changes in the footprint of the LSA, specific extraction processes, and the nature and design of collection systems. Additionally, there have been past remediation efforts at the LIU. Three major historical PLS releases have occurred; in 1985, 1988, and 2007. The first two releases affected Tributary 1, and the most recent Tributary 2. Remediation and monitoring related to the 2007 spill were addressed under DP-376. A major remediation effort was conducted for the 2007 event in which a large amount of sediment and surface water was removed. Contamination from this event reportedly did not extend beyond the confluence of Tributary 1 and 2. The RI report ([Arcadis, 2012], Section 2.8.8) contains further details.

2.3 Exposure Models

2.3.1 Description

Exposure models are qualitative and quantitative (i.e., equations) means to 'translate' EPCs into estimates of receptor exposure. These estimates are in turn combined with estimates of COI toxicity to estimate risks.

In general, exposure models incorporate assumptions regarding the following:

- Types of receptors
- Characteristics and behavior of those receptors
- Likely areas where receptors will live, work, recreate, etc.; and how these intersect with the spatial extent of contamination

These assumptions are collectively termed 'scenarios'. Typically, such scenarios are based upon current and likely future land use. At an operating site such as the LIU, future land use is difficult to predict, as this is highly dependent upon the market for the mine's product, general economic factors, population pressures, changing demographics, and other factors. Therefore, the LIU HHRA will only evaluate the most likely scenarios, based upon consultation with NMED and Chino, current use, and observation of land use in surrounding areas.

In the type of HHRA conducted here, exposure is typically estimated for a hypothetical receptor under 'reasonable maximum exposure' (RME) conditions [EPA, 1992b]. The intent of the RME concept is to ensure that it is likely that typical exposures and risks will be overestimated, as opposed to underestimated; but not to evaluate absolute worst-case conditions.

According to EPA [EPA, 1992b]:

> *The RME, which is defined as the highest exposure that could reasonably be expected to occur for a given exposure pathway at a site, is intended to account for both uncertainty in the contaminant concentration and variability in exposure parameters (e.g., exposure frequency, averaging time).*

There are two ways that RME is typically defined:

A. By defining exposure scenarios that include assumptions and activities that would result in a comparatively large degree of exposure. Typically this involves allowing receptors to live on the site, as well as other activities

B. By including a number of conservative assumptions (e.g., 95th percentiles of population distributions) in exposure models (e.g., a receptor who breathes at a high rate, drinks water at a high rate, etc.), along with upper-bound estimates for EPCs

Both of these RME 'methods' are employed in the HHRA. In the case of B above, qualitative judgments are made regarding upper-bound estimates to result in RME, as opposed to worst-case, estimates. Additionally, deterministic HHRAs (e.g., previous HHRAs at the Chino site) often employ 'central tendency', 'average', or 'typical' assumptions as a point of comparison. This is addressed in the LIU HHRA as germane to A above by including current use (e.g., ranching). In the case of B above, the difficulty lies in determining the proper combination of values that actually result in an 'average' exposure. Additionally, the degree of conservatism associated with deterministic RME exposure model results may be unknown, so comparisons between an 'average' estimate and a RME estimate do not provide an accurate estimate of the degree of bias associated with the RME estimate. Basically, there is a large degree of confidence that a RME estimate is conservative in terms of typical risks, but the degree of conservatism is unknown.

Therefore, for the purpose of this HHRA, an alternative approach is proposed. RME exposures are estimated in the usual fashion. If unacceptable risks are found in the Tier II assessment, and/or if the involved parties are not comfortable with the degree of uncertainty associated with risk estimates, then a probabilistic analysis [EPA, 2001;Cullen, A. and Frey, H. C., 1999] can be conducted only for those COPCs and exposure pathways that are problematic. This will allow a much more detailed and accurate representation of uncertainties than is provided by a comparison between RME and 'average' or 'central tendency' exposure/risk estimates. Additionally, the probabilistic analysis will allow determination of the degree of conservatism associated with RME estimates; e.g., whether a RME estimate represent a 90th percentile, a 99th percentile, and so on. If appropriate, this will provide the involved parties the best and most complete information for decision-making.

2.3.2 Exposure Scenarios

The most likely generic scenarios, based upon current land use, previous HHRAs (i.e., for the Hanover Whitewater Creek (HWC) and Smelter Tailings Soil (STS) IUs; see

[Neptune, 2008;Gradient, 2008]), a tour of the overall site, and discussions with Chino and NMED, include the following:

A. Present and future commercial ranching
B. Present trespassing on Chino property
C. Future residential development
D. Future recreation (e.g., hiking)
E. Future construction work

The land in the vicinity of Tributaries 1 and 2 is presently owned by Chino Mines Company and leased for cattle grazing. Access to this area for the general public via Highway 152 and other roads is feasible, but limited. Based upon interviews with Chino staff, there appears to be no current recreational use, because the area is fenced and use beyond the approved ranching would be considered trespassing. There are no current residences on the property, although the nearest is only 1 km from the eastern LIU boundary. Evaluation of potential future land use follows precedent set in the HWCIU and STSIUs HHRAs [Neptune, 2008;Gradient, 2008]. In both these IUs, areas that are presently owned by Chino but could feasibly support future development were evaluated under a range of land use options. Future residential development is possible at the LIU, although the likelihood of this is difficult to judge at this time.

Some exposure-related activities may be predominantly associated with specific geomorphic (i.e., land and geology) features and locations. For example, residences may be limited by terrain, proximity to roads, and other considerations. Therefore, a distinction is made between exposures over the entire IU (including both uplands and tributaries) versus exposures to only upland areas.

Receptors may be exposed to COIs via ingestion, inhalation, and dermal (skin) absorption. Ingestion may include dust/soil and water. Inhalation involves breathing in dust, and dermal absorption involves COIs being deposited and absorbed into the skin.

It is assumed here that potential residential groundwater use in upland areas would be unlikely to be affected by site impacts. Regardless, groundwater is addressed under the regulatory structure of the DP. COIs are monitored in a number of wells, and any exceedences of groundwater criteria trigger regulatory action under the DP. As this regulatory situation is likely to continue for the foreseeable future, groundwater is not explicitly addressed in this HHRA. Some receptors might drink surface water at the LIU. Surface water is generally ephemeral in the tributaries, but pools can persist. Ranchers and recreationists, for example, could occasionally drink this water; assuming treatment for microorganisms. The RI [Arcadis, 2012] found that there were no exceedences of regulatory drinking water criteria (e.g., Maximum Contaminant Levels) for some COIs at the site, but the HHRA is not using these screening criteria as they are not necessarily risk-based. Surface water is therefore retained for the HHRA in terms of ingestion. It is unlikely that dermal contact would be an important exposure pathway given intermittent and brief exposures.

In the HWCIU and STSIU HHRAs [Neptune, 2008;Gradient, 2008], food pathways (e.g., home-grown produce, chickens, beef, etc.) were evaluated. In the case of the LIU, NMED has determined that modeling potential exposures related to home-raised foods,

game and ranching would provide limited information for risk management in the LIU. NMED has decided not to pursue the foodstuff pathways in the LIU HHRA because of the low likelihood that future residents would engage in extensive agricultural activities or hunt extensive site-related game, and because cultivation of produce will likely require amendments for productive garden soil due to the poor quality of existing soil.

A beef ingestion exposure pathway may be of concern at the LIU in a situation where cattle may be slaughtered at the time they are grazing locally. In this case, receptors could have access to meat that could form a significant portion of their diet over an extensive exposure period. By contrast, commercial cattle are commonly slaughtered after they have been moved from pasture to a feedlot and the meat is processed and sold commercially to a broad market. According to information collected by Chino from the current commercial ranching leaseholder, local consumption of beef from animals grazed in the area of the LIU is unlikely, and this pathway has thus not been evaluated.

Table 1 describes specific exposure scenarios evaluated in the LIU HHRA:

Table 1: Exposure Scenarios

Scenario	Receptor Type	Age	Examples of Activities	Location of Activities	Exposure routes	Notes
A: Ranching	Ranch-hand	Adult	Herding cattle, mending fences	Upland and tributaries	Dust inhalation, soil ingestion, dermal absorption, surface water ingestion	*Present and future* Assumed to live off the LIU site, and not to consume product
B: Trespassing	Local resident	Adult	Walking, shooting	Upland and tributaries	Dust inhalation, soil ingestion, dermal absorption, surface water ingestion	*Present* Assumes that young children would not trespass
C: Residence	Family	Adult, child (0-6 years)	Living in house, playing in yard, walking on property	Upland	Dust inhalation, soil ingestion, dermal absorption	*Future* Location assumes negligible probability that a house would be built in a tributary due to flooding risk, and that minimal exposure to tributaries exists
D: Recreation	Local resident	Adult	Hiking	Upland and tributaries	Dust inhalation, soil ingestion, dermal absorption, surface water ingestion	*Future* Assumes that young children (0-6 years) would not recreate in the area
E: Construction	Local resident	Adult	Digging, operating machinery, construction work	Upland	Dust inhalation, soil ingestion, dermal absorption	*Future* Location assumes low probability that a house (or other building) would be built in a tributary due to flooding risk

Note that children are only evaluated in the residential Scenario C. "Children" are defined as from birth to 6 years, and specifically ages 3 to 6 in terms of soil ingestion [EPA, 2011], as this age group tends to engage in hand-to-mouth activities that result in more soil ingestion. This essentially defines this age group as a special population, as soil ingestion often is a driving factor in soil-related risk. Otherwise, "adult" exposure variable values are generally defined here as those relevant to ages 16 and older. Ages in-between are not evaluated explicitly, but this is not expected to affect the estimates of risk appreciably. The selection of adults rather than older children as receptors in the trespassing and recreational scenarios will be re-examined if the risk assessment results are close to thresholds of concern.

3 Data Evaluation for Constituents of Interest

3.1 Constituent of Interest Evaluation

It is important that all site-related COIs are identified, and that the concentrations are accurately quantified [EPA, 1994a]. Constituents carried forward to Tier II of the HHRA are identified as COPCs via the Tier I screening assessment. Details of statistical analyses performed for Tier II are described in that section.

Data collected in the RI and historical data (summarized in [Arcadis, 2012]) are used in the HHRA. The RI and previous studies utilized standard quality assurance/quality control procedures in collection and analysis of data. Data tables were provided by Chino and ARCADIS in report and Excel spreadsheet formats. There are four types of data collected in the RI and previous studies that are germane to the HHRA: Surface soil (0-1"); shallow soil (0-6"); sediment (generally 0-6"); and, surface water. Non-detected values are not addressed in the Tier I screening as maximum detected values are assessed, but non-detects are addressed in Tier II. Note that not all sediment data are relevant due to historical releases and subsequent remediation that occurred. Table 4-9 in the RI [Arcadis, 2012] presents the sampling locations and dates reflective of "current conditions" (e.g., Table 4-9 suggests that sediment data collected for Tributary 2 in early 2008 are not appropriate due to the recency of the major PLS spill). Data that are not representative of current conditions are not considered in the HHRA. However, this summary table does not present information for all COIs, so information for additional COIs via the primary sources is identified.

Surface water values used here are the "dissolved" fraction, as it is reasonable to assume that most users would filter water before drinking it. Where possible, these conform to the approximate locations and dates as presented in Table 4-11 in the RI report [Arcadis, 2012] as most relevant to current conditions. "Flood" values (e.g., in Table 2-3 in the RI report) are not used here, as persistent pools are of interest.

3.2 Background Concentrations

The issue of 'background' is complex, but important. Any mine site has highly mineralized deposits in its undisturbed state (i.e., pre-mining). Thus, environmental concentrations of metals may be high compared to other locations, even if there is no

present or past mining activity. Comparisons of metal concentrations between mining-affected areas and non-affected areas (i.e., background or reference areas) are therefore difficult. The responsible party at a contaminated site (in this case, Chino) is not responsible for remediating areas of high background concentrations that have not been affected by the LIU stockpiles. At the LIU, upwind reference areas to the northwest and southwest of the Lampbright stockpile operations were chosen based upon previous investigations. The reference areas are assumed to be largely upwind of the stockpile, and are assumed to be representative of the mineralized geology within the LIU [Arcadis, 2012] (Figure 3-4). It was found that areas to the north of the stockpiles were highly mineralized. For this reason, NMED has chosen not to conduct *a priori* comparisons between site-related COI concentrations and LIU reference area concentrations for the purpose of screening mining-related COIs. Rather, the Tier II HHRA estimates COPC risks for the LIU site data, and then examines the LIU reference area data and the reference area data from the STSIU [Gradient, 2008]. The STSIU reference area was relatively non-mineralized in nature and may represent less-mineralized areas of the LIU. It is beyond the scope of the HHRA to incorporate extensive geological investigations; although this information may be used in risk management decisions.

3.3 Data Analysis

Neptune has assessed the adequacy of site and reference area data based upon completeness, comparability, and representativeness to make a final determination of whether the data are adequate to characterize the exposure areas at the LIU and are appropriate for use in the HHRA in terms of screening, estimating EPCs, and other purposes. Although the RI conducted a number of statistical analyses, additional analyses are necessary for the purpose of the HHRA. Details of statistical analyses performed for Tier II are described in that section. Briefly, data analysis activities begin with exploratory data analysis using descriptive statistics and box-and-whisker plots. Graphical analysis is often the most important step in presenting and interpreting the data. These graphs help direct and interpret the ensuing statistical analyses by providing initial evidence of the likely results. The exact nature of statistical testing that is conducted to confirm the findings of the exploratory data analysis depends upon the data and the distributional forms they support.

3.4 Further Data Collection and Analysis

NMED recommended further sampling to characterize arsenic associated with one sampling location, as described in the RI report [Arcadis, 2012]. Further recommendations are made in the context of the Uncertainty Assessment and Conclusions sections.

4 Quality Assurance/Quality Control

The HHRA relies upon numerous data and information sources, and employs a number of assumptions and calculations. No primary data were collected. All provided/published primary site and reference area data were checked and confirmed in multiple sources

(reports and spreadsheets) where possible. The following were checked by at least one internal reviewer:

- COI/COPC data entry
- Statistical data entry and output
- Exposure variable assumption sources and entry
- Toxicity values sources and entry
- Workbook calculations
- Report content

5 Tier I Screening Human Health Risk Assessment

5.1 Exposure Assessment

5.1.1 Estimation and Use of Exposure Point Concentrations

The Tier I screening analysis employed in the HHRA uses maximum detected concentrations of all COIs for EPCs in exposure models detailed in Appendix I. Table 2 below summarizes data requirements for the Tier I screening. Different receptor types in the defined scenarios will be exposed to different proportions of soil vs. sediment, depending upon activities and specific areas. For the purposes of Tier I screening, the assumptions in the table are applied. These are professional judgments, based upon the following:

- Current ranching (A) largely occurs in upland areas (90% surface soil, 10% sediment), and this situation is likely to continue into the future
- Current trespassing (B) would likely occur in more 'interesting' areas closer to the mine's activities, and thus largely in upland areas (90% surface soil, 10% sediment)
- Future residence (C) would involve only upland areas, and would not involve substantial contact with sediment (100% surface soil)
- Future recreation (D) could occur in any area, but is more likely than other scenarios to involve sediment (50% soil, 50% sediment)
- Future construction (E) would only occur in upland areas, and would involve deeper layers of soil (100% shallow soil)

These assumptions are subject to uncertainty, and the impact is discussed in the Uncertainty Assessment section.

Table 2: Tier I Analysis Data Requirements

Scenario	Receptor Type	Location of Activities	Exposure routes	Relevant Media: Maximum COI Concentrations for Screening	Assumptions
A: Commercial ranching	Ranch-hand	Upland and tributaries	Dust inhalation, soil ingestion, dermal absorption, surface water ingestion	Surface soil (0-1") and sediment (0-6") Surface water	Contact with surface layer of soil (90%) and sediment (10%)
B: Trespassing	Local resident	Upland and tributaries	Dust inhalation, soil ingestion, dermal absorption, surface water ingestion	Surface soil (0-1") and sediment (0-6") Surface water	Contact with surface layer of soil (90%) and sediment (10%)
C: Residence	Family	Upland	Dust inhalation, soil ingestion, dermal absorption	Surface soil (0-1"): dust inhalation, soil ingestion, dermal absorption from soil	100% of activities involving surface soil layer
D: Recreation	Local resident	Upland and tributaries	Dust inhalation, soil ingestion, dermal absorption, surface water ingestion	Surface soil (0-1") and sediment (0-6") Surface water	Contact with surface soil (50%) and sediment (50%)
E: Construction	Local resident	Upland	Dust inhalation, soil ingestion, dermal absorption	Shallow soil (0-6")	100% of activities involving surface and deeper soil layers

In addition to the RI report [Arcadis, 2012], several previous studies provide relevant data [Chino, 1995;SRK, 2008;Golder, 2008;Golder, 2010]. The RI report summarizes the results of these studies as germane to the LIU. The types of data and constraints were previously described.

Table 3 below presents EPCs (maximums) for the Tier I screening. All of the data collected in the RI are referenced to the RI report, but were confirmed from data tables (Excel) provided by Arcadis and Chino.

Table 3: Tier I Maximum Detected Relevant Values for Constituents of Interest

COI	Surface Soil (mg/kg 0-1")	Primary Reference	Table/ Sample ID in RI [Arcadis, 2012]	Shallow Soil (mg/kg 0-6")	Primary Reference	Table/ Sample ID in RI [Arcadis, 2012]	Sediment (mg/kg 0-6")	Primary Reference	Table/ Sample ID in RI [Arcadis, 2012]	Surface Water (mg/L)	Table/ Sample ID in RI [Arcadis, 2012]	Primary Reference
Aluminum	29400	[Chino, 1995]	Table 2-1 (2005)	29600	[Arcadis, 2012]	Table 4-5 (L-20)	19500	[Chino, 1995]	Table 2-2 (2214)	0.11	Table 2-10 (2410)	[Golder, 2008]
Arsenic	29	[Arcadis, 2012]	Table 4-1 (L-20)	36	[Arcadis, 2012]	Table 4-5 (L-20)	6.6	[Arcadis, 2012]	Table 4-8 (65+40)	U	–	–
Barium	486	[Arcadis, 2012]	Table 4-1 (L-13)	566	[Arcadis, 2012]	Table 4-5 (L-18)	164	[SRK, 2008]	Table 2-17 (LAMP-BRIGHT TRIB-1-2(1.5-2)	0.14	Table 2-10 (376-05-04)	[Golder, 2008]
Beryllium	1.4	[Arcadis, 2012]	Table 4-1 (L-19)	1.5	[Arcadis, 2012]	Table 4-5 (L-04)	1.1	[Arcadis, 2012]	Table 4-8 (65+40)	U	–	–
Boron	11	[Arcadis, 2012]	Table 4-1 (L-19, 20)	13	[Arcadis, 2012]	Table 4-5 (L-19)	2.3	[Arcadis, 2012]	Table 4-8 (65+40)	0.15	Table 2-10 (LB7S)	[Golder, 2008]
Cadmium	2.0	[Chino, 1995]	Table 2-1 (2005)	1.7	[Arcadis, 2012]	Table 4-5 (L-18)	0.57	[Arcadis, 2001]	Table 2-4 (ERA34-2)	0.0042	Table 2-12 (LB-12)	[Golder, 2010]
Chromium total	99	[SRK, 2008]	Table 2-16 (SS102)	63	[Arcadis, 2012]	Table 4-5 (L-20)	86	[Golder, 2008]	Table 2-11 (376-05-04)	0.0023	Table 4-10 (Trib 2A-SW)	[Arcadis, 2012]
Chromium III	85	Assumes 6:1 III/VI ratio	--	54	Assumes 6:1 III/VI ratio	–	74	Assumes 6:1 III/VI ratio	–	0.0020	Assumes 6:1 III/VI ratio	–
Chromium VI	14	Assumes 6:1 III/VI ratio	--	9.0	Assumes 6:1 III/VI ratio	–	12	Assumes 6:1 III/VI ratio	–	0.00033	Assumes 6:1 III/VI ratio	–
Cobalt	24	[SRK, 2008]	Table 2-16 (SS102)	13	[Arcadis, 2012]	Table 4-5 (L-08)	22	[Chino, 1995]	Table 2-2 (2214)	0.027	Table 2-12 (LBT-11)	[Golder, 2010]
Copper	614	[Arcadis, 2012]	Table 4-1 (L-07)	319	[Arcadis, 2012]	Table 4-5 (L-08)	721	[Chino, 1995]	Table 2-2 (2214)	0.036	Table 2-10 (LBT1-BF1)	[Golder, 2008]
Iron	34200	[SRK, 2008]	Table 2-16 (SS102)	27500	[Arcadis, 2012]	Table 4-5 (L-19)	25400	[SRK, 2008]	Table 2-17 (LAMP-BRIGHT TRIB-1-2(1.5-2)	0.094	Table 2-10 (2410)	[Golder, 2008]

Continued Next Page

COI	Surface Soil (mg/kg 0-1")	Primary Reference	Table/ Sample ID in RI [Arcadis, 2012]	Shallow Soil (mg/kg 0-6")	Primary Reference	Table/ Sample ID in RI [Arcadis, 2012]	Sediment (mg/kg 0-6")	Primary Reference	Table/ Sample ID in RI [Arcadis, 2012]	Surface Water (mg/L)	Table/ Sample ID in RI [Arcadis, 2012]	Primary Reference
Lead	67	[Arcadis, 2012]	Table 4-1 (L-21)	107	[Arcadis, 2012]	Table 4-5 (L-05)	37	[Golder, 2008]	Table 2-11 (LBT143-BF1)	0.024	Table 2-12 (LBT-11)	[Golder, 2010]
Manganese	1440	[Arcadis, 2012]	Table 4-1 (L-21)	841	[Arcadis, 2012]	Table 4-5 (L-21)	1050	[Chino, 1995]	Table 2-2 (2214)	1.9	Table 2-10 (376-05-04)	[Golder, 2008]
Mercury	NA	--	--	NA	--	--	0.04	[Arcadis, 2001]	Table 2-4 (ERA34-2)	NA	--	--
Molybdenum	23	[Arcadis, 2012]	Table 4-1 (L-08)	8.9	[Arcadis, 2012]	Table 4-5 (L-08)	10	[Chino, 1995]	Table 2-2 (2214)	0.038	Table 2-10 (376-05-04)	[Golder, 2008]
Nickel	36	[Chino, 1995]	Table 2-1 (2005)	40	[Arcadis, 2012]	Table 4-5 (L-20)	13	[Chino, 1995]	Table 2-2 (2214)	0.011	Table 2-12 (LBT-11)	[Golder, 2010]
Selenium	1.5	[Chino, 1995]	Table 2-1 (2005)	0.90	[Arcadis, 2012]	Table 4-5 (L-01)	0.34	[Golder, 2008]	Table 2-14 (T2S7)	0.00070	Table 4-10 (156+50-SW))	[Arcadis, 2012]
Silver	NA	--	--	NA	--	--	0.31	[Chino, 1995]	Table 2-2 (2224)	NA	--	--
Thallium	NA	--	--	NA	--		0.13	[Arcadis, 2001]	Table 2-4 (ERA34-1)	NA	--	--
Vanadium	125	[SRK, 2008]	Table 2-16 (SS102)	65	[Arcadis, 2012]	Table 4-5 (L-05)	38	[SRK, 2008]	Table 2-17 (LAMPBRIGHT TRIB-1-1(0-0.5)	0.0062	Table 2-10 (LB7S)	[Golder, 2008]
Zinc	886	[Chino, 1995]	Table 2-1 (2001)	125	[Arcadis, 2012]	Table 4-5 (L-08)	208	[Chino, 1995]	Table 2-2 (2214)	1.1	Table 2-12 (LBT-11)	[Golder, 2010]

NA: not analyzed; U: not detected

5.1.2 Exposure Variable Assumptions

5.1.2.1 Introduction

The Tier I HHRA for the LIU incorporates numerous assumptions for present and future receptor exposures. The equations used in both Tiers of the HHRA are presented in Appendix I.

Exposure variable assumptions (RME values) for Tier I of the HHRA include largely conservative values for the sake of 'protective' screening of COIs. Exceptions are noted below.

All exposure variable values are presented in Table 4, along with references. Explanations are presented below. Note that some of the assumptions may vary from previous HHRAs. This is largely due to recent updated information in the latest EPA Exposure Factors Handbook [EPA, 2011], published in 2011. This source documents the best and most recent information on many exposure variables, and is used as a primary source. Where possible, the recommended values presented in the Executive Summary of that report (Table ES-1) are used here. However, this source does not contain information on all of the exposure variables of interest; thus, other sources are employed when necessary, including professional judgment.

Note that the term "soil" is used here to represent both upland soil and sediment. In general, "children" are defined where possible to represent the approximate age range of EPA's concern in terms of soil ingestion; i.e., 3 to 6 years of age. This is not always possible due to availability of data, thus exceptions are noted.

Variables are presented in approximate alphabetical order below.

5.1.2.2 Averaging Time

Averaging time (AT; in yr) is employed in the case of carcinogens to 'average' exposure over a lifetime, as there is assumed to be a lifetime risk of cancer upon exposure. A value of 78 yr is used for all scenarios. According to EPA [EPA, 2011], this is the mean (i.e., arithmetic mean, or average) lifespan (males plus females) as represented in (Table ES-1). For non-carcinogenic toxicants, the AT is equivalent to exposure duration (see below), as lifetime exposures are not assumed to be of interest in terms of toxicity (i.e., toxic effects do not persist throughout the lifetime of the receptor).

5.1.2.3 Bioavailability Fractions

COIs can absorb to soil or dust particles, resulting in less ability for these compounds to be absorbed into the bloodstream. Relative bioavailability is the amount of a compound that is able to be absorbed into the bloodstream via various soil related exposure routes, compared to soluble forms in food or water (as employed in derivation of toxicity values). Many metal compounds have low bioavailability in the gastrointestinal tract, and less via the skin. Bioavailability is not typically addressed for inhaled COIs, as the toxicity values are directly based upon inhalation studies.

Relative bioavailability fractions (BFs; dimensionless) are measured using *in vitro* (laboratory assays) or *in vivo* (animal) studies. Note that if BFs are used for some COIs

and not for others in a HHRA, then it is essentially assumed that the BF is zero for the COIs that do not have assigned BFs. Thus, if BFs are to be used at all, then BFs must be applied to all COIs.

Values of dermal and ingestion BFs used in the Tier I calculations are provided in Table 5. Relative bioavailability is defined as the bioavailability of the COI in soil divided by the bioavailability of the COI in the critical study related to the oral toxicity criterion. EPA does not publish a summary of ingestion (gastrointestinal) BFs. Ingestion BFs were used inconsistently in previous Chino HHRAs. NMED [NMED, 2012] publishes ingestion BF information for some COIs, but the sources and derivation are unclear. A relative ingestion bioavailability of 1.0 is therefore protectively used for all COIs in the Tier I ingestion calculations, as ingestion often dominates dermal exposure to metals.

EPA and NMED do not publish a summary of a wide range of dermal BFs (BF_derm). However, values of BF_derm for most of the COIs are published in the Ontario Ministry of the Environment's soil standard development guidelines [Ontario MOE, 2011]. BF_derm may also be calculated as the absolute dermal absorption fraction (see Table 2.25 in [Ontario MOE, 2011], and Exhibit 3-4 in [EPA, 2004]) divided by the bioavailability of the COI in the critical study related to the oral toxicity criterion ([EPA, 2004]; Exhibit 4-1). For the Tier I calculations, the larger of the calculated values for BF_derm and those published in the Ontario MOE guidance [Ontario MOE, 2011] is used. In a few cases these values exceeded 1.0; thus these BF values are set to 1.0.

5.1.2.4 Body Weight

Exposures are adjusted by body weight (BW; in kg), as some other variables change depending upon the size of individual, and toxicity is typically body mass dependent. According to EPA [EPA, 2011], a mean BW for adults (BW_a) over 21 is 80 kg (Table ES-1). For children (BW_c) from age 0 to 6 years in Scenario C, the time-weighted mean of the mean values for these age strata (15 kg) is used. For the first year, the time-weighted infant body weight is 7.8 kg. This value is then combined with body weights for ages 1 – <2 yr (11.4 kg), 2 – <3 yr (13.8 kg), and 3 – <6 yr (18.6 kg) for a time-weighted result of 14.8 kg, rounded to 15 kg.

5.1.2.5 Dermal Surface Area

The dermal surface area (DSA; in cm^2) is an estimate of the exposed area of skin that would be available for contact with soil and dust (as much of the body is typically covered by clothes). Mean values are appropriate, as skin surface area is correlated with body weight. For adults, only the hands and arms are assumed to be exposed. The DSAs of arms and hands for adult males over 21 in EPA [EPA, 2011] (Table ES-1) are 3140 cm^2 and 1070 cm^2, respectively. For children, exposed skin is assumed to be available for arms, hands, legs, and feet. Skin surface area for a child age 3 – <6 yr is protectively used in the Tier I calculations. The mean DSAs from EPA [EPA, 2011] (Table ES-1) are; arms (1060 cm^2), hands (370 cm^2), legs (1950 cm^2), and feet (490 cm^2).

5.1.2.6 Dermal Soil Adherence Factor

The dermal soil adherence factor (DSAF; in mg/cm^2) is a measure of how much soil is retained by the skin during particular activities. The assumption here is that exposure

events (as represented in EPA [EPA, 2004].) are integrated over the period of a day, consistent with later EPA guidance [EPA, 2011] that no longer represents DSAF on a per-event basis. Note that it is possible to have a high degree of adherence when activities are conducted in mud or wet sediment; however, assuming that all activities will take place in such media is unrealistic.

For adults, the mean soil adherence for construction activities (the highest adherence) from EPA [EPA, 2011] (Table ES-1) for arms (0.1859 mg/cm^2) and hands (0.2763 mg/cm^2) is used. For children, mean soil adherence for "activities with soil" is used for arms (0.046 mg/cm^2), hands (0.17 mg/cm^2), legs (0.051 mg/cm^2), and feet (0.20 mg/cm^2).

5.1.2.7 Exposure Duration

RME exposure durations (EDs; in yr) are employed here, per EPA "standard default factors" [EPA, 1991]. For adults, an ED of 25 yr for Scenarios A and E, 30 yr is used for Scenarios B and D, and 24 yr (i.e., 30 minus 6 yrs for children) for Scenario C. For children, an ED of 6 yr is used to match the age range of concern in Scenario C.

5.1.2.8 Exposure Frequency

RME exposure frequencies (EFs; in d/yr) represent the amount of time that receptors are expected to spend conducting activities in each of the scenarios. The EFs for Scenarios A and E represent EPA standard default values for RME workers; i.e., 250 d/yr [EPA, 1991]. For Scenarios B and D, a professional judgment of 50 d/yr is assumed, based upon once per wk, 50 wks per yr. For Scenario C, the standard default residential EF of 350 d/yr [EPA, 1991] is used for both adults and children.

5.1.2.9 Exposure Time

RME exposure times (ETs; in hr/d) represent the amount of time that receptors are expected to spend 'on site'. A typical working day is assumed to be 8 hrs, and defines ETs for adults in Scenarios A and E. It is also assumed that trespassers in Scenario B and recreationists in Scenario D would not spend more than 8 hr/d in the affected area. Thus, the same ET is protectively used for adults in these scenarios. For Scenario C, it is assumed that adults will spend 8 hr/d away from home, thus ET_a is 16 hr/d. This estimate is consistent with the EPA-estimated sum of mean time spent indoors (Table 16-1; approximately 900 minutes) and outdoors (Table 16-22; approximately 140 minutes) at a residence [EPA, 2011]. Mean estimates of time indoors at a residence from birth to age 6 years vary between approximately 65 and 75% [EPA, 2011] (Table 16-1). For Tier I, it is protectively assumed that children in this scenario will spend 24 hr/d at home.

5.1.2.10 Fraction of Ingestion/Dermal Contact Associated with Site

For soil/dust ingestion and dermal exposure to soil, it is useful to include a fractional factor (FS; dimensionless) that accounts for exposure for some fraction of time off-site; i.e., to noncontaminated soil and dust. For the purpose of Tier I screening, however, this factor will be set to 1.0; thus assuming all soil contact is with site soils.

5.1.2.11 Ingestion Rate of Soil and Dust

Ingestion rates of soil and dust (IR_s; in mg/d) are difficult to quantify, and thus subject to a large degree of uncertainty. The rates assumed here are standard EPA RME values. It

is beyond the scope of the Tier I assessment to characterize the degree of uncertainty, but this will be examined further in Tier II if soil/dust ingestion is a driving exposure pathway in terms of risk.

Adults are assumed to ingest soil and dust at the same rate across scenarios. EPA [EPA, 2011] does not provide an upper-percentile value for adults. The value in Table 1 (100 mg/d) represents the "central tendency" estimate for ages 6 to 21, which is more conservative than the "adult" estimate (50 mg/d) in EPA [EPA, 2011] (Table ES-1). The value for children (200 mg/d) is an "upper percentile" estimate for children aged 3 to 6 [EPA, 2011] (Table ES-1).

5.1.2.12 Ingestion Rate of Water

Ingestion rate of water (IR_w; in L/d) is evaluated for Scenarios A, B, and D; and thus only adults are evaluated. It is assumed that these receptors would only drink from surface water pools on an intermittent basis, and residents and construction workers would not drink from such sources at all.

As drinking water from such sources would be intermittent, it would be inappropriate to assume upper-percentile values for IR_w. Additionally, a distinction should be made between routine community sources of water and other sources. Therefore, a 50^{th} percentile rate (0.22 L/d) for "other sources" is assumed for consumers-only greater than 21 years, as provided in EPA [EPA, 2011] (Table 3-35).

Table 4: Tier I Exposure Assumptions

		A Ranching		*B Trespass*		*C Residence*		*D Recreation*		*E Construction*	
		Value	Reference	Value	Reference	Value	Reference	Value	Reference	Value	Reference
Variable	Units										
AT: carcinogens	yr	78	[EPA, 2011], Table ES-1	78	[EPA, 2011], Table ES-1	78	[EPA, 2011], Table ES-1	78	[EPA, 2011], Table ES-1	78	[EPA, 2011], Table ES-1
AT: noncarcinogens	yr	Equal to exposure duration	--	Equal to exposure duration	--	Equal to exposure duration	--	Equal to exposure duration	--	Equal to exposure duration	--
BW_a	kg	80	[EPA, 2011], Table ES-1	80	[EPA, 2011], Table ES-1	80	[EPA, 2011], Table ES-1	80	[EPA, 2011], Table ES-1	80	[EPA, 2011], Table ES-1
BW_c	kg	--	--	--	--	15	[EPA, 2011], Table ES-1	--	--	--	--
DSA_a	cm^2	3140; 1070	[EPA, 2011], Table ES-1 (see text)	3140; 1070	[EPA, 2011], Table ES-1 (see text)	3140; 1070	[EPA, 2011], Table ES-1 (see text)	3140; 1070	[EPA, 2011], Table ES-1 (see text)	3140; 1070	[EPA, 2011], Table ES-1 (see text)
DSAF_a	mg/cm^2	0.1859; 0.2763	[EPA, 2011], Table ES-1 (see text)	0.1859; 0.2763	[EPA, 2011], Table ES-1 (see text)	0.1859; 0.2763	[EPA, 2011], Table ES-1 (see text)	0.1859; 0.2763	[EPA, 2011], Table ES-1 (see text)	0.1859; 0.2763	[EPA, 2011], Table ES-1 (see text)
DSA_c	cm^2	1060; 370; 1950; 490	[EPA, 2011], Table ES-1 (see text)	1060; 370; 1950; 490	[EPA, 2011], Table ES-1 (see text)	1060; 370; 1950; 490	[EPA, 2011], Table ES-1 (see text)	1060; 370; 1950; 490	[EPA, 2011], Table ES-1 (see text)	1060; 370; 1950; 490	[EPA, 2011], Table ES-1 (see text)
DSAF_c	mg/cm^2	0.046; 0.17; 0.051; 0.2	[EPA, 2011], Table ES-1 (see text)	0.046; 0.17; 0.051; 0.2	[EPA, 2011], Table ES-1 (see text)	0.046; 0.17; 0.051; 0.2	[EPA, 2011], Table ES-1 (see text)	0.046; 0.17; 0.051; 0.2	[EPA, 2011], Table ES-1 (see text)	0.046; 0.17; 0.051; 0.2	[EPA, 2011], Table ES-1 (see text)
ED_a	yr	25	[EPA, 1991]	30	[EPA, 1991]	24	[EPA, 1991]	30	[EPA, 1991]	1	[EPA, 1991]
ED_c	yr	--	--	--	--	6	[EPA, 1991]	--	--	--	--
EF_a	d/yr	250	[EPA, 1991]	50	See text	350	[EPA, 1991]	50	See text	250	[EPA, 1991]
EF_c	d/yr	--	--	--	--	350	[EPA, 1991]	--	--	--	--
ET_a	hr/d	8	See text	4	See text	16	See text	8	See text	8	See text
ET_c	hr/d	--	--	--	--	24	See text	--	--	--	--
IR_s_a	mg/d	100	[EPA, 2011], Table ES-1	100	[EPA, 2011], Table ES-1	100	[EPA, 2011], Table ES-1	100	[EPA, 2011], Table ES-1	100	[EPA, 2011], Table ES-1
IR_s_c	mg/d	--	--	--	--	200	[EPA, 2011], Table ES-1	--	--	--	--
IR_w	L/d	0.22	[EPA, 2011], Table 3-35	0.22	[EPA, 2011], Table 3-35	--	--	0.22	[EPA, 2011], Table 3-35	--	--

--: not applicable

Table 5: Tier I Relative Bioavailability Factors

COI	Dermal Absorption Fraction [Ontario MOE, 2011]	Gastrointestinal Absorption Fraction [EPA, 2004]	Relative Bioavailability; Soil, Dermal [Ontario MOE, 2011]	*Relative Dermal Bioavailability* [1]	*Relative Oral Bioavailability*
Aluminum	0.01	1.0	NA	0.01	1.0
Arsenic	0.04[2]	1.0	0.04	0.04	1.0
Barium	0.01	0.07	0.1	0.14	1.0
Beryllium	0.01	0.007	0.1	1.0	1.0
Boron	0.01	1.0	0.01	0.01	1.0
Cadmium	0.001	0.025	0.01	0.04	1.0
Chromium III[3]	0.04	0.013	0.1	1.0	1.0
Chromium VI	0.04	0.025	0.1	1.0	1.0
Cobalt	0.01	1.0	0.01	0.01	1.0
Copper	0.01	1.0	0.06	0.06	1.0
Iron	0.01	1.0	NA	0.01	1.0
Manganese	0.01	0.04	NA	0.25	1.0
Mercury	0.1	0.07	0.1	1.0	1.0
Molybdenum	0.01	1.0	0.01	0.01	1.0
Nickel	0.04	0.04	0.2	1.0	1.0
Selenium	0.01	1.0	NA	0.01	1.0
Silver	0.01	0.04	NA	0.25	1.0
Thallium	0.01	1.0	NA	0.01	1.0
Vanadium	0.01	0.026	NA	0.38	1.0
Zinc	0.01	1.0	NA	0.01	1.0

NA: not available. Relative bioavailability is defined as the bioavailability of the COI in soil divided by the bioavailability of the COI in the critical toxicity study. All BFs are dimensionless. All oral BFs are defined as 1.0 for Tier I protective screening, due to lack of available summary information.

1. The maximum of (Dermal Absorption Fraction / GI Absorption Fraction) or Relative Bioavailability; Soil, Dermal. This value of BF_derm is used in Tier I (to a maximum of 1.0.)
2. EPA [EPA, 2004] publishes a value of 0.03 for arsenic; however, to be consistent and conservative the Ontario value is applied here.
3. As total chromium.

5.2 Toxicity Assessment

5.2.1 Introduction

Regulatory toxicant-specific toxicity values for evaluating non-cancer (a wide range of chronic toxic effects) and cancer health endpoints are referred to as reference doses (RfD) and slope factors (SF), respectively; as well as their inhalation counterparts (see below). These are essentially 'conversion factors' applied to intake estimates in order to determine whether unacceptable risks exist at a site. Tier I of the HHRA assumes the following, in accordance with EPA policy:

- The only carcinogenic COIs are those that EPA designates as such
- All cancers are 'equal' in importance
- Non-carcinogenic toxicity is typically secondary in importance to carcinogenicity
- There is no uncertainty in regulatory toxicity values; thus, they essentially amount to policy criteria

The primary source of regulatory toxicity values is EPA's Integrated Risk Information System database (IRIS; [EPA, 2012a]). Toxicity values published in IRIS have gone through peer-review and EPA-consensus-review processes. The second tier of toxicity criteria are the provisional peer reviewed toxicity values (PPRTVs) published by the National Center for Environmental Assessment in EPA's Office of Research and Development [EPA, 2012b]. These values are developed on a chemical-specific basis when requested by EPA's Superfund program. The third tier of references include values published in EPA's Health Effects Assessment Summary Tables (HEAST; [EPA, 1997]) and other sources such as California EPA and the US Agency for Toxic Substances and Disease Registry. The toxicity values presented in Table 6 below are consistent with the most recent NMED soil screening guidance [NMED, 2012] and the EPA RSL summary tables [EPA, 2012c].

There are many important considerations and assumptions inherent in these toxicity values that should be evaluated before making risk management decisions. For example, some metals are considered essential elements. While this will not be considered quantitatively in the HHRA, essentiality will be discussed in the context of uncertainty associated with any potential risks associated with such metals.

For Tier I these toxicity values are only presented for screening purposes; thus, extensive discussion of toxicity is not presented. Toxicity for COPCs in Tier II is discussed more extensively. The "critical effects" for non-carcinogens are presented in Table 7, as these are important in terms of potential additivity across COIs; i.e. if both toxicants A and B cause kidney toxicity, then their effects may be considered together, whereas a liver toxicant C may not be considered along with A and B. It is possible that any or all of the COIs may have multiple toxic effects that may interact, but assessment of such interactions would be highly complex and would introduce a level of uncertainty that would be difficult to characterize. Thus, for the purpose of the HHRA, it is assumed that EPA's judgment of critical effects is the only criterion by which COIs would be jointly

considered. This assumption will be discussed in the Uncertainty Assessment section of the Tier II HHRA.

Table 8 provides a preliminary indication of toxicants whose effects may be additive. Toxicity for COPCs evaluated in Tier II is discussed in more detail in that section.

5.2.2 Oral Reference Doses and Reference Concentrations

The toxicity value used to evaluate non-cancer, systemic (i.e., not limited to the site of absorption) health effects related to long-term exposures is the chronic reference dose (RfD; in mg/kg-d). The chronic RfD is an estimate of daily exposure likely to be without appreciable risk of adverse effects for exposure of several years or longer [EPA, 1989].

The general model of toxicity for non-carcinogenic effects is that there is range of exposure from zero to some 'threshold' in which exposure can be tolerated without an adverse effect. An oral RfD represents an estimate of this threshold and is expressed as rate of exposure (normalized for body weight) with the same units as intake (i.e., mg/kg-d). Intake is then divided by the RfD; if the ratio is greater than 1.0, then toxic effects may occur (the probability of such effects is not evaluated). This model of toxicity is reflected in the averaging time for non-carcinogenic effects, which is equivalent to the exposure duration. The toxic effect is assumed to occur only when exposure exceeds a threshold and not to occur when exposure is less than the threshold or at some time following termination of the exposure.

RfDs are derived by EPA using human dose-response data from occupational or epidemiological studies, if available. In some cases, clinical case reports have been used. If human data are unavailable, dose-response information from animal studies may be employed. EPA will preferentially base a RfD upon the highest dose level not associated with adverse effects (the no-observable-adverse-effects-level, or NOAEL). If such a value was not identified in the literature, the lowest-observable-adverse-effects-level (LOAEL) is generally used as the basis of the RfD. In practice, EPA will generally first identify the critical study and adverse effect for a chemical from a review of the available toxicological data. Once these are specified the NOAEL or LOAEL is identified.

The RfD is then calculated from the NOAEL or LOAEL using uncertainty factors (UFs) to adjust (downward; i.e., toward more conservative) the NOAEL or LOAEL to a chronic RfD. UFs do not address uncertainty per se; rather, they add layers of protectiveness to the available information. UFs may relate to potential variability in sensitivity in the human population, to interspecies variability between humans and test animals, to inadequate dosing periods in a critical study, or to use of a LOAEL instead of a NOAEL. A 'modifying factor' is sometimes employed to adjust these values further. So, in practice, a LOAEL may be lowered several orders of magnitude with multiple UFs and other factors to result in a protective RfD.

Inhalation of chronic toxicants is addressed somewhat differently. EPA [EPA, 2009] again estimates reference concentrations (RfCs; with units of mg/m^3) by extrapolating from human or animal studies, but these values implicitly incorporate assumptions regarding body weight and inhalation rate. Thus, exposures to inhaled toxicants are estimated by comparing a chronic level of exposure (in mg/m^3) with the RfC.

RfDs and RfCs for screening of COIs are presented in Table 6.

5.2.3 Oral Slope Factors and Inhalation Unit Risks

The toxicity value used to evaluate carcinogenic health effects is the slope factor (SF; in $(mg/kg\text{-}d)^{-1}$). A SF is a quantitative relationship between dose and carcinogenic response. This relationship is assumed to be linear (i.e., the greater the dose, the greater the risk of cancer) with no threshold (i.e., any amount of exposure to the carcinogen can result in excess risk). The majority of SFs are based upon carcinogenic effects observed at high dose rates (e.g., in animal experiments or occupational studies) that have been extrapolated to lower doses using a linearized multistage model. The SF is usually an upper-bound estimate (although there are exceptions) of the lifetime probability of developing cancer associated with exposure to a specific quantity of a potential carcinogen [EPA, 1989]; i.e., incremental lifetime cancer risk (ILCR). The SF is expressed as cancer risk per unit intake [risk/ (mg/ kg-d)], or $(mg/\ kg\text{-}d)^{-1}$. Because there may be a decades-long latency period between exposure and effect, effects are averaged over an entire lifetime. ILCR is a concept routinely used by EPA in environmental HHRA [EPA, 1989]. "Incremental' is defined as the risk associated with a specific exposure that is increased over all-cause cancer risk. This is approximately 1 in 2 (45%) for males, and 1 in 3 (38%) over an average lifetime, and the lifetime risk of dying from cancer is approximately 20% [ACS, 2012]. Thus, the ILCR is the site-related risk over-and-above this.

Similar to RfCs, inhalation of carcinogens is addressed differently. Toxicity values are provided in IRIS and HEAST as inhalation unit risks (URs; in $(\mu g/m^3)^{-1}$). Thus, exposures to inhaled toxicants are estimated by comparing a chronic level of exposure (in $\mu g/m^3$) with the UR.

When a chemical is thought to cause mutations in genetic material (i.e., a mutagenic mode-of-action, or MOA), EPA assumes that exposure to such chemicals may pose particularly high cancer risk to infants and young children. The only COI that this potentially applies to here is CrVI. EPA provides guidance [EPA, 2005b] for adjusting cancer potency estimates for childhood exposures for carcinogens that have such a MOA. For the purpose of the HHRA, detailed age-specific exposure variable values are not employed. Rather, generic 'children'-related values are used.

SFs and URs for screening of COIs are presented in Table 6.

5.2.4 Lead Risk

Lead risk is addressed differently from other metals by EPA, as it exhibits complex dynamics in the human body. EPA recommends a residential screening level for lead in soil of 400 mg/kg, derived using a biokinetic model. The 400 mg/kg screening level was developed such that a typical child would have no more than a 5% chance of having a blood lead level exceeding 10 µg/dl, a level thought to be associated with developmental health effects in children [EPA, 1994b]. Site-related residential exposures contributing to the 400 mg/kg screening level include soil ingestion from a yard and indoor ingestion of house dust contaminated with soil. In addition to these site-related exposures, the 400

mg/kg screening level incorporates background levels of lead exposure from non-site related sources including ambient air, drinking water, and diet. These background exposures were defined using "national averages, where suitable, or typical values" [EPA, 1994b]. For the purpose of the LIU HHRA, the 400 mg/kg value has been adopted for assessing potential risks to children in the residential Scenario C. No soil or sediment values in the LIU exceed this criterion; therefore lead will not be addressed further in the HHRA.

Table 6: Tier I Toxicity Values

COI	Oral RfD		Inhalation RfC		Oral SF		Inhalation UR	
	(mg/kg-d)	Reference	(mg/m^3)	Reference	$(mg/kg\text{-}d)^{-1}$	Reference	$(\mu g/m^3)^{-1}$	Reference
Aluminum	1.0E+00	[EPA, 2012b]	5.0E-03	[EPA, 2012b]	NA	NA	NA	NA
Arsenic	3.0E-04	[EPA, 2012a]	1.5E-05	[CalEPA, 2008b]	1.5E+00	[EPA, 2012a]	4.3E-03	[EPA, 2012a]
Barium	2.0E-01	[EPA, 2012a]	5.0E-04	[EPA, 1997]	NA	NA	NA	NA
Beryllium	2.0E-03	[EPA, 2012a]	2.0E-05	[EPA, 2012a]	NA	NA	2.4E-03	[EPA, 2012a]
Boron	2.0E-01	[EPA, 2012a]	2.0E-02	[EPA, 1997]	NA	NA	NA	NA
Cadmium[1]	1.0E-03 (diet) 5.0E-04 (water)	[EPA, 2012a]	2.0E-05	[CalEPA, 2008b]	NA	NA	1.8E-03	[EPA, 2012a]
Chromium (III)	1.5E+00	[EPA, 2012a]	NA	NA	NA	NA	NA	NA
Chromium (VI)[2]	3.0E-03	[EPA, 2012a]	1.0E-04	[EPA, 2012a]	5.0E-01	[NJDEP, 2009]	8.4E-02	[EPA, 2012a]
Cobalt	3.0E-04	[EPA, 2012b]	6.0E-06	[EPA, 2012b]	NA	NA	9.0E-03	[EPA, 2012b]
Copper[3]	4.0E-02	[EPA, 1997]	2.4E-02	[Gradient, 2008]	NA	NA	NA	NA
Iron	7.0E-01	[EPA, 2012b]	NA	NA	NA	NA	NA	NA
Manganese[4]	1.4E-01 (diet) 2.4E-02 (other)	[EPA, 2012a]	5.0E-05	[EPA, 2012a]	NA	NA	NA	NA
Mercury[5]	3.0E-04	[EPA, 2012a]	3.0E-05	[CalEPA, 2008b]	NA	NA	NA	NA
Molybdenum	5.0E-03	[EPA, 2012a]	NA	NA	NA	NA	NA	NA
Nickel[6]	2.0E-02	[EPA, 2012a]	9.0E-05	[ATSDR, 2005]	NA	NA	2.6E-04	[CalEPA, 1991]
Selenium	5.0E-03	[EPA, 2012a]	2.0E-02	[CalEPA, 2008b]	NA	NA	NA	NA
Silver	5.0E-03	[EPA, 2012a]	NA	NA	NA	NA	NA	NA
Thallium[7]	1.0E-05	[EPA, 2012b]	NA	NA	NA	NA	NA	NA
Vanadium[8]	5.0E-03	[EPA, 2012c]	NA	NA	NA	NA	NA	NA
Zinc	3.0E-01	[EPA, 2012a]	NA	NA	NA	NA	NA	NA

Continued Next Page

NA: not available or applicable

1. According to RSL guidance [EPA, 2012c]: "food" is for food and soil use; "water" is for water only. Further, the cadmium RfDs in IRIS are based upon the same study. The food RfD incorporates a 2.5% absorption adjustment; the water RfD incorporates a 5% absorption adjustment. For another medium such as soil, the risk assessor should choose the number whose absorption factor most closely matches the expected conditions at the site. For example, if the expected absorption of cadmium from soil is 3%, the food-based number would be a good approximation. In most cases, the expected absorption is unknown and the RfD for food should be used for soil screening without making any changes to the value."
2. The inhalation unit risk value of 1.2E-02 per $\mu g/m^3$ in IRIS [EPA, 2012a] assumes the ratio of CrIII to CrVI in air is 6:1 (as the critical study involved a mixture). The value of 8.4E-02 per $\mu g/m^3$ is adjusted by a factor of 7 for CrVI only. The RfC for CrVI is for particulates.
3. The RfD is calculated from drinking water standard of 1.3 mg/L, per HEAST [EPA, 1997] and RSL guidance [EPA, 2012c] via multiplication by 2 L/d and division by 70 kg. The RfC was developed by Gradient, based upon occupational limits [Gradient, 2008].
4. According to RSL guidance [EPA, 2012c]: "the IRIS RfD includes manganese from all sources, including diet. The explanatory text in IRIS recommends using a modifying factor of 3 when calculating risks associated with non-food sources, and the SL table follows this recommendation. IRIS also recommends subtracting dietary exposure (default assumption in this case is 5 mg). Thus, the IRIS RfD has been lowered by a factor of 2 x 3, or 6. The table now reflects manganese for "non-food" sources".
5. As mercuric chloride and other salts.
6. As nickel soluble salts. The RfC in the RSL table [EPA, 2012c] is based upon an Agency for Toxic Substances and Disease Registry Minimum Risk Level.
7. Value for thallium soluble salts is based upon information in an appendix to the PPRTV documentation [EPA, 2012b]; see http://hhpprtv.ornl.gov/issue_papers/ThalliumandCompounds.pdf.
8. Value based upon vanadium as opposed to vanadium pentoxide. The IRIS value is for vanadium pentoxide; however, this form is not likely to be a major constituent in mining-related releases. The RSL table adjusts this value for vanadium.

Table 7: Tier I Constituent of Interest Critical Toxic Effects

Non-Carcinogenic Chronic Toxicity				
COI	**Oral RfD**	**Reference**	**Inhalation RfC**	**Reference**
Aluminum	Neurological, psychomotor and cognitive impairment	[EPA, 2012b]	Neurotoxicity	[EPA, 2012b]
Arsenic	Hyperpigmentation, keratosis (skin) and possible vascular complications	[EPA, 2012a]	Development; cardiovascular system; nervous system; lung; skin	[CalEPA, 2008b]
Barium	Nephropathy	[EPA, 2012a]	Fetotoxicity	[EPA, 1997]
Beryllium	Intestinal lesions	[EPA, 2012a]	Sensitization, chronic beryllium disease (respiratory toxicity)	[EPA, 2012a]
Boron	Developmental (decreased fetal weight)	[EPA, 2012a]	Respiratory irritation, bronchitis	[EPA, 1997]
Cadmium	Protenuria (nephrotoxicity)	[EPA, 2012a]	Kidney; respiratory system	[CalEPA, 2008b]
Chromium (III)	No effects	[EPA, 2012a]	NA	NA
Chromium (VI)	No effects	[EPA, 2012a]	Lactate dehydrogenase in bronchioalveolar lavage fluid (lung toxicity)	[EPA, 2012a]
Cobalt	Central nervous system effects	[EPA, 2012b]	Respiratory irritation, decreased lung function	[EPA, 2012b]
Copper	Gastrointestinal irritation	[EPA, 1997]	Respiratory irritation	[Gradient, 2008]
Iron	Gastrointestinal toxicity	[EPA, 2012b]	NA	NA
Manganese	Central nervous system effects	[EPA, 2012a]	Impairment of neuro-behavioral function	[EPA, 2012a]
Mercury	Autoimmune effects	[EPA, 2012a]	Nervous system	[CalEPA, 2008b]
Molybdenum	Increased uric acid levels (nephrotoxicity)	[EPA, 2012a]	NA	NA
Nickel	Decreased body and organ weights	[EPA, 2012a]	Lung inflammation	[ATSDR, 2005]
Selenium	Clinical selenosis (thickened/brittle nails, hair/nail loss, lowered hemoglobin levels, mottled teeth, skin lesions and CNS abnormalities)	[EPA, 2012a]	Alimentary system; cardiovascular system; nervous system	[CalEPA, 2008b]
Silver	Argyria (permanent bluish-gray discoloration of the skin)	[EPA, 2012a]	NA	NA
Thallium	Hair follicle atrophy	[EPA, 2012b]	NA	NA
Vanadium	Kidney histopathology	[EPA, 2012b]	NA	NA
Zinc	Decreases erythrocyte Zn superoxide dismutase (ESOD) activity	[EPA, 2012a]	NA	NA
Cancer Sites				
COI	**Oral SF**	**Reference**	**Inhalation UR**	**Reference**
Arsenic	Skin	[EPA, 2012a]	Lung	[EPA, 2012a]
Beryllium	NA	NA	Lung	[EPA, 2012a]
Cadmium	NA	NA	Lung, trachea, bronchus	[EPA, 2012a]
Chromium VI	Intestine	[NJDEP, 2009]	Lung	[EPA, 2012a]
Cobalt	NA	NA	Lung, bronchus	[EPA, 2012b]
Nickel	NA	NA	Lung	[CalEPA, 1991]

NA: not available

Table 8: Tier I Constituents of Interest with Similar Toxic Effects

Oral Route Toxicity	COIs	Inhalation Route Toxicity	COIs
Gastrointestinal irritation	Copper, iron	*Respiratory irritation*	Boron, cadmium, chromium VI, cobalt, copper, nickel
Neurotoxicity	Aluminum, cobalt, manganese	*Neurotoxicity*	Aluminum, arsenic, manganese, mercury, selenium
Nephrotoxicity	Barium, cadmium, molybdenum, vanadium	*Cardiovascular effects*	Arsenic, selenium
		Developmental toxicity	Arsenic, barium
		Lung cancer	Arsenic, beryllium, cadmium, chromium VI, nickel

5.3 Risk Characterization

5.3.1 Overview

In risk characterization, site-related COI exposures and toxicity values are combined to produce estimates of incremental lifetime cancer risk (ILCR) and non-carcinogenic hazard. These estimates are then compared with 'acceptable' levels, as determined by regulatory guidance, precedent, and discussion among involved parties (see below) to determine COPCs for Tier II.

5.3.2 Estimation of Non-Carcinogenic Hazards

Chronic RfDs specific for COIs and intake routes are used to convert estimated daily intake over an exposure period to a HQ. A HQ does not reflect the probability of an effect occurring. However, larger values of HQ can be associated with potentially increased severity of effects. The equation for calculating the HQ is:

$$HQ = \frac{Intake}{RfD}$$

where,

HQ = hazard quotient
Intake = chronic daily intake (mg/kg-d)
RfD = reference dose (mg/kg-d)

The RfD is assumed to be linearly related to HQ in this equation. HQs above 1.0 (i.e., the estimate intake level exceeds the RfD) are of potential concern, consistent with EPA and

NMED guidance. The potential for additive non-carcinogenic effects across two or more COIs is evaluated in the HHRA only in cases where the critical toxic effects of the COIs are similar. The sum of two or more HQ values is referred to as a Hazard Index (HI). A HI value exceeding 1.0 may be of concern even if the HQs for all individual COIs are below 1.0, but only if the individual COIs have similar toxicological endpoints (see Table 8).

HQs are summed across both route of intake and exposure pathways for a given scenario. The potential additivity of pathways, and particularly whether a RME exposure could occur for two or more pathways simultaneously for an individual, is evaluated before pathway hazards are simply summed.

The HQ or HI value that is generally indicative of the potential for adverse health effects is 1.0. Information provided in the Toxicity and Uncertainty Assessment sections regarding the confidence and potential biases associated with HQ or HI estimates will be used to inform the involved parties in determining an appropriate decision if HQ or HI values are above 1.0.

5.3.3 Estimation of Incremental Lifetime Cancer Risks

SFs specific for COIs and exposure routes are used to convert estimated daily intake over an exposure period to ILCR, as:

$$ILCR = Intake \times SF$$

where,

ILCR	=	lifetime incremental cancer risk (dimensionless)
Intake	=	chronic daily intake (mg/kg-d)
SF	=	slope factor $(mg/kg\text{-}d)^{-1}$

ILCR estimates are calculated for individual COIs. Typically these are summed across both route of intake and exposure pathways for a given scenario. However, the potential additivity of pathways, and particularly whether a RME could occur for two or more pathways simultaneously for an individual, is evaluated before pathway risks are summed.

ILCRs across individual COIs have generally been summed to estimate a total ILCR, in accordance with EPA guidance [EPA, 1989]. However, there are a number of issues associated with this policy. 'Cancer' is not one disease, but hundreds; each having a unique clinical profile, mode or mechanism of action, and natural history (e.g., liver cancer is a very different disease than skin cancer). Only 50% of cancers are fatal (over a lifetime; [ACS, 2012]), and many are benign. Additionally, there are differences in the derivation and level of confidence associated with individual SFs and URs. As indicated in Table 7, the lungs are a common site of cancer for all the carcinogenic COIs for the

inhalation exposure route. Therefore, inhalation UR results are summed in this risk assessment. No common sites of cancer exist for the two oral route carcinogens (arsenic and CrVI), and the scientific and statistical bases of the SFs for these chemicals are very different. It is difficult to interpret the addition of such disparate carcinogenic risks. The oral cancer ILCRs for these chemicals are thus evaluated independently in this assessment and discussed in the Toxicity Assessment and Uncertainty Assessment sections of the HHRA.

The final ILCR that may be acceptable for risk management decisions will be determined by the involved parties. The ILCR *de minimus* (i.e., minimal or not measurable in a public health study) range of 1E-04 (i.e., 1×10^{-4}) to 1E-06 (0.0001 to 0.000001) described in the National Contingency Plan [EPA, 1990] has been routinely used by involved parties as a decision aid (note that EPA guidance [EPA, 1989] specifies presentation of ILCRs with one significant figure). NMED has defined 1E-05 (0.00001) as a target for development of its Soil Screening Levels (SSLs) [NMED, 2012]. For context, the total lifetime risk of cancer to a male receptor exposed to a COI that is associated with a 1E-05 ILCR would be approximately 0.45 plus 0.00001, or 0.45001.

Only risk-relevant COIs screened in the Tier I assessment are carried forward to the Tier II assessment. For Tier I, a 1E-05 ILCR is used as a screening target level.

5.3.4 Results

5.3.4.1 Overview

Results of the Tier I screening risk characterization are presented below. Results are presented for each of the scenarios, as well as relevant exposure pathways.

5.3.4.2 Hazard Quotient Results

Results are presented in Tables 9 through 13 as individual COI HQs, plus COIs are combined as appropriate in terms of toxic effects (see Table 8). In all cases, "NA" in tables refers to a lack of relevant COI data (either not detected or not analyzed) or missing toxicity values; thus no HQ is estimated. "NR" means "not relevant".

Bold values exceed a HQ of 0.1, and ***bold/italic*** values exceed a HQ of 1.0. Only COI with HQs exceeding 0.1 are summed, and then only if toxicologically relevant (per Table 8). Only individual COIs with HQs greater than 1.0 or toxicologically relevant combinations of COIs (HIs) greater than 1.0 are carried forward into Tier II. Note that these results are for screening only and should not be used for informing risk management decisions.

Scenario C (residence) is the only scenario evaluated that included children. Children tend to exceed chronic toxicity criteria (e.g., greater than a HQ of 1.0) to a greater degree than adults, due to lower body weight, higher soil contact, and other considerations. In all cases, the estimated HQs for children exceeded those of adults. Thus, for the purpose of Tier I screening, only children will be represented here for Scenario C. Interpretation of results follows the tables.

Table 9: Tier I Scenario A (Commercial Ranching) Hazard Quotients

COI	Soil Ingestion	Dermal Absorption	Soil Ingestion + Dermal Absorption	Dust Inhalation	Surface Water Ingestion	*Total*	Retain?
Aluminum	2.4E-02	2.1E-03	2.6E-02	9.7E-03	2.1E-04	3.6E-02	NO
Arsenic	7.5E-02	2.6E-02	1.0E-01	3.0E-03	NA	1.0E-01	NO
Barium	1.9E-03	2.4E-03	4.4E-03	1.5E-03	1.3E-03	7.2E-03	NO
Beryllium	5.9E-04	5.2E-03	5.7E-03	1.2E-04	NA	5.9E-03	NO
Boron	4.3E-05	3.8E-06	4.7E-05	8.5E-07	1.4E-03	1.5E-03	NO
Cadmium	1.6E-03	5.6E-04	2.1E-03	1.6E-04	1.6E-02	1.8E-02	NO
Chromium III	4.8E-05	4.2E-04	4.7E-04	NA.	NA	4.7E-04	NO
Chromium VI	4.0E-03	3.5E-02	3.9E-02	2.4E-04	1.4E-03	4.1E-02	NO
Cobalt	6.8E-02	6.0E-03	7.4E-02	6.8E-03	**1.7E-01**	**2.5E-01**	NO
Copper	1.3E-02	7.1E-03	2.0E-02	4.4E-05	1.7E-03	2.2E-02	NO
Iron	4.1E-02	3.6E-03	4.4E-02	NA	2.5E-04	4.5E-02	NO
Manganese	5.0E-02	**1.1E-01**	**1.6E-01**	4.8E-02	**1.5E-01**	**3.6E-01**	NO
Mercury	1.1E-05	1.0E-04	1.1E-04	2.3E-07	NA	1.1E-04	NO
Molybdenum	3.8E-03	3.3E-04	4.1E-03	n.a.	1.4E-02	1.8E-02	NO
Nickel	1.4E-03	1.3E-02	1.4E-02	6.3E-04	1.0E-03	1.6E-02	NO
Selenium	2.4E-04	2.1E-05	2.6E-04	1.2E-07	2.6E-04	5.2E-04	NO
Silver	5.3E-06	1.2E-05	1.7E-05	NA	NA	1.7E-05	NO
Thallium	1.1E-03	9.8E-05	1.2E-03	NA	NA	1.2E-03	NO
Vanadium	2.0E-02	6.7E-02	8.7E-02	NA	2.3E-03	9.0E-02	NO
Zinc	2.3E-03	2.1E-04	2.5E-03	NA	6.8E-03	9.4E-03	NO
TOTAL gastrointestinal irritation (copper and iron)	5.4E-02	1.1E-02	6.5E-02	NR	1.9E-03	6.7E-02	NO
TOTAL neurotoxicity (aluminum, arsenic, cobalt, manganese, mercury, selenium)	**2.2E-01**	**1.4E-01**	**3.6E-01**	6.7E-02	**3.2E-01**	**7.5E-01**	NO
TOTAL nephrotoxicity (barium, cadmium, molybdenum, vanadium)	2.7E-02	7.1E-02	9.8E-02	1.7E-03	3.4E-02	**1.3E-01**	NO
TOTAL cardiovascular effects (arsenic, selenium)	7.5E-02	2.6E-02	1.0E-01	3.0E-03	2.6E-04	1.0E-01	NO

NA: lack of relevant COI data (either not detected or not analyzed) or missing toxicity values. NR: not relevant. **Bold** values exceed HQ of 0.1, and ***bold/italic*** values exceed HQ of 1.0. Only COIs with HQs exceeding 0.1 are summed for screening purposes, and then only if toxicologically relevant.

Table 10: Tier I Scenario B (Trespassing) Hazard Quotients

COI	Soil Ingestion	Dermal Absorption	Soil Ingestion + Dermal Absorption	Dust Inhalation	Surface Water Ingestion	*Total*	Retain?
Aluminum	4.9E-03	4.3E-04	5.3E-03	9.7E-04	4.1E-05	6.3E-03	NO
Arsenic	1.5E-02	5.2E-03	2.0E-02	3.0E-03	NA	2.0E-02	NO
Barium	3.9E-04	4.9E-04	8.8E-04	1.5E-03	2.6E-04	1.3E-03	NO
Beryllium	1.2E-04	1.0E-03	1.1E-03	1.2E-04	NA	1.2E-03	NO
Boron	8.6E-06	7.6E-07	9.4E-06	8.5E-07	2.8E-04	2.9E-04	NO
Cadmium	3.2E-04	1.1E-04	4.3E-04	1.6E-04	3.2E-03	3.6E-03	NO
Chromium III	9.6E-06	8.4E-05	9.4E-05	NA	NA	9.4E-05	NO
Chromium VI	7.9E-04	7.0E-03	7.8E-03	2.4E-04	2.9E-04	8.1E-03	NO
Cobalt	1.4E-02	1.2E-03	1.5E-02	6.8E-03	3.4E-02	4.9E-02	NO
Copper	2.7E-03	1.4E-03	4.1E-03	4.4E-05	3.4E-04	4.4E-03	NO
Iron	8.2E-03	7.2E-04	8.9E-03	NA	5.1E-05	8.9E-03	NO
Manganese	1.0E-02	2.2E-02	3.2E-02	4.8E-03	3.0E-02	6.6E-02	NO
Mercury	2.3E-06	2.0E-05	2.2E-05	2.3E-07	NA	2.2E-05	NO
Molybdenum	7.5E-04	6.6E-05	8.2E-04	NA	2.9E-03	3.7E-03	NO
Nickel	2.8E-04	2.5E-03	2.8E-03	6.3E-04	2.1E-04	3.1E-03	NO
Selenium	4.7E-05	4.2E-06	5.2E-05	1.2E-07	5.3E-05	1.0E-04	NO
Silver	1.1E-06	2.3E-06	3.4E-06	NA	NA	3.4E-06	NO
Thallium	2.2E-04	2.0E-05	2.4E-04	NA	NA	2.4E-04	NO
Vanadium	4.0E-03	1.3E-02	1.7E-02	NA	4.7E-04	1.8E-02	NO
Zinc	4.7E-04	4.1E-05	5.1E-04	NA.	1.4E-03	1.9E-03	NO
TOTAL gastrointestinal irritation (copper and iron)	1.1E-02	2.1E-03	1.3E-02	NR	3.9E-04	1.3E-02	NO
TOTAL neurotoxicity (aluminum, arsenic, cobalt, manganese, mercury, selenium)	4.3E-02	2.9E-02	7.2E-02	6.7E-03	6.4E-02	**1.4E-01**	NO
TOTAL nephrotoxicity (barium, cadmium, molybdenum, vanadium)	5.4E-03	1.4E-02	2.0E-02	1.7E-04	6.8E-03	2.7E-02	NO
TOTAL cardiovascular effects (arsenic, selenium)	1.5E-02	5.3E-03	2.0E-02	3.0E-04	5.3E-05	2.1E-02	NO

NA: lack of relevant COI data (either not detected or not analyzed) or missing toxicity values. NR: not relevant. **Bold** values exceed HQ of 0.1, and ***bold/italic*** values exceed HQ of 1.0. Only COIs with HQs exceeding 0.1 are summed for screening purposes, and then only if toxicologically relevant.

Table 11: Tier I Scenario C (Residence) Hazard Quotients (child)

COI	Soil Ingestion	Dermal Absorption	Soil Ingestion + Dermal Absorption	Dust Inhalation	Surface Water Ingestion	Total	Retain?
Aluminum	**3.8E-01**	5.8E-03	**3.8E-01**	4.2E-02	NR	**4.2E-01**	**YES**[1]
Arsenic	***1.2E+00***	7.5E-02	***1.3E+00***	1.3E-02	NR	***1.3E+00***	***YES***
Barium	3.1E-02	6.9E-03	3.8E-02	6.9E-03	NR	4.5E-02	NO
Beryllium	8.9E-03	1.4E-02	2.3E-02	5.0E-04	NR	2.3E-02	NO
Boron	7.0E-04	1.1E-05	7.1E-04	3.9E-06	NR	7.1E-04	NO
Cadmium	2.6E-02	1.6E-03	2.7E-02	7.1E-04	NR	2.8E-02	NO
Chromium III	7.2E-04	1.1E-03	1.8E-03	NA	NR	1.8E-03	NO
Chromium VI	6.0E-02	9.3E-02	**1.5E-01**	1.0E-03	NR	**1.5E-01**	NO
Cobalt	**1.0E+00**	1.6E-02	**1.0E+00**	2.9E-02	NR	***1.1E+00***	***YES***
Copper	**2.0E-01**	1.8E-02	**2.1E-01**	1.8E-04	NR	**2.1E-01**	NO
Iron	**6.2E-01**	9.7E-03	**6.3E-01**	NA	NR	**6.3E-01**	NO
Manganese	**7.7E-01**	**3.0E-01**	***1.1E+00***	**2.1E-01**	NR	***1.3E+00***	***YES***
Mercury	NA	NA	NA	NA	NR	NA	NO
Molybdenum	6.0E-02	9.2E-04	6.1E-02	NA	NR	6.1E-02	NO
Nickel	2.3E-02	3.5E-02	5.8E-02	2.8E-03	NR	6.1E-02	NO
Selenium	3.8E-03	5.9E-05	3.9E-03	5.4E-07	NR	3.9E-03	NO
Silver	NA	NA	NA	NA	NR	NA	NO
Thallium	NA	NA	NA	NA	NR	NA	NO
Vanadium	**3.2E-01**	**1.9E-01**	**5.1E-01**	NA	NR	**5.1E-01**	NO
Zinc	3.8E-02	5.8E-04	3.8E-02	NA	NR	3.8E-02	NO
TOTAL gastrointestinal irritation (copper and iron)	**8.2E-01**	2.8E-02	**8.5E-01**	NR	NR	**8.5E-01**	NO
TOTAL neurotoxicity (aluminum, arsenic, cobalt, manganese, mercury, selenium)	***3.4E+00***	**3.9E-01**	***3.8E+00***	**2.9E-01**	NR	***4.1E+00***	***YES*** **(aluminum, arsenic, cobalt, manganese)**
TOTAL nephrotoxicity (barium, cadmium, molybdenum, vanadium)	**4.4E-01**	**2.0E-01**	**6.4E-01**	7.7E-03	NR	**6.4E-01**	NO
TOTAL cardiovascular effects (arsenic, selenium)	***1.2E+00***	7.5E-02	***1.3E+00***	1.3E-02	NR	***1.3E+00***	***YES*** **(arsenic)**

NA: lack of relevant COI data (either not detected or not analyzed) or missing toxicity values. NR: not relevant. **Bold** values exceed HQ of 0.1, and ***bold/italic*** values exceed HQ of 1.0. Only COIs with HQs exceeding 0.1 are summed, and then only if toxicologically relevant.
1: Retained on the basis of potential additive effects.

Table 12: Tier I Scenario D (Recreation) Hazard Quotients

COI	Soil Ingestion	Dermal Absorption	Soil Ingestion + Dermal Absorption	Dust Inhalation	Surface Water Ingestion	*Total*	Retain?
Aluminum	4.2E-03	3.7E-04	4.6E-03	1.7E-03	4.1E-05	6.3E-03	NO
Arsenic	1.0E-02	3.5E-03	1.3E-02	3.0E-03	NA	1.4E-02	NO
Barium	2.8E-04	3.5E-04	6.3E-04	1.5E-03	2.6E-04	1.1E-03	NO
Beryllium	1.1E-04	9.4E-04	1.0E-03	1.2E-04	NA	1.1E-03	NO
Boron	5.7E-06	5.0E-07	6.1E-06	8.5E-07	2.8E-04	2.9E-04	NO
Cadmium	2.2E-04	7.7E-05	3.0E-04	1.6E-04	3.2E-03	3.5E-03	NO
Chromium III	9.1E-06	8.0E-05	8.9E-05	NA	NA	8.9E-05	NO
Chromium VI	7.5E-04	6.6E-03	7.4E-03	2.4E-04	2.9E-04	7.7E-03	NO
Cobalt	1.3E-02	1.2E-03	1.4E-02	6.8E-03	3.4E-02	5.0E-02	NO
Copper	2.9E-03	1.5E-03	4.4E-03	4.4E-05	3.4E-04	4.7E-03	NO
Iron	7.3E-03	6.4E-04	7.9E-03	NA	5.1E-05	8.0E-03	NO
Manganese	8.9E-03	2.0E-02	2.8E-02	8.5E-03	3.0E-02	6.7E-02	NO
Mercury	1.1E-05	1.0E-04	1.1E-04	2.3E-07	NA	1.1E-04	NO
Molybdenum	5.8E-04	5.1E-05	6.3E-04	NA	2.9E-03	3.5E-03	NO
Nickel	2.1E-04	1.8E-03	2.0E-03	6.3E-04	2.1E-04	2.3E-03	NO
Selenium	3.2E-05	2.8E-06	3.4E-05	1.2E-07	5.3E-05	8.7E-05	NO
Silver	5.3E-06	1.2E-05	1.7E-05	NA	NA	1.7E-05	NO
Thallium	1.1E-03	9.8E-05	1.2E-03	NA	NA	1.2E-03	NO
Vanadium	2.8E-03	9.4E-03	1.2E-02	NA	4.7E-04	1.3E-02	NO
Zinc	3.1E-04	2.7E-05	3.4E-04	NA	1.4E-03	1.7E-03	NO
TOTAL gastrointestinal irritation (copper and iron)	1.0E-02	2.2E-03	1.2E-02	NR	3.9E-04	1.3E-02	NO
TOTAL neurotoxicity (aluminum, arsenic, cobalt, manganese, mercury, selenium)	3.6E-02	2.5E-02	6.1E-02	1.2E-02	6.4E-02	**1.4E-01**	NO
TOTAL nephrotoxicity (barium, cadmium, molybdenum, vanadium)	3.9E-03	9.9E-03	1.4E-02	2.4E-04	6.8E-03	2.1E-02	NO
TOTAL cardiovascular effects (arsenic, selenium)	1.0E-02	3.5E-03	1.4E-02	4.0E-04	5.3E-05	1.4E-02	NO

NA: lack of relevant COI data (either not detected or not analyzed) or missing toxicity values. NR: not relevant. **Bold** values exceed HQ of 0.1, and ***bold/italic*** values exceed HQ of 1.0. Only COIs with HQs exceeding 0.1 are summed, and then only if toxicologically relevant.

Table 13: Tier I Scenario E (Construction) Hazard Quotients

COI	Soil Ingestion	Dermal Absorption	Soil Ingestion + Dermal Absorption	Dust Inhalation	Surface Water Ingestion	*Total*	Retain?
Aluminum	2.5E-02	2.2E-03	2.7E-02	**5.3E-01**	NR	**5.5E-01**	**YES[1]**
Arsenic	8.1E-02	2.8E-02	**1.1E-01**	**1.7E-01**	NR	**2.8E-01**	**YES[1]**
Barium	2.1E-03	2.6E-03	4.7E-03	8.7E-02	NR	9.2E-02	NO
Beryllium	6.0E-04	5.3E-03	5.9E-03	6.3E-03	NR	1.2E-02	NO
Boron	4.7E-05	4.1E-06	5.1E-05	4.9E-05	NR	1.0E-04	NO
Cadmium	1.7E-03	6.0E-04	2.3E-03	8.9E-03	NR	1.1E-02	NO
Chromium III	4.8E-05	4.3E-04	4.7E-04	NA	NR	4.7E-04	NO
Chromium VI	4.0E-03	3.5E-02	3.9E-02	1.3E-02	NR	5.2E-02	NO
Cobalt	6.8E-02	6.0E-03	7.5E-02	**3.6E-01**	NR	**4.3E-01**	**YES[1]**
Copper	1.3E-02	6.9E-03	2.0E-02	2.3E-03	NR	2.2E-02	NO
Iron	4.2E-02	3.7E-03	4.6E-02	NA	NR	4.6E-02	NO
Manganese	5.1E-02	**1.1E-01**	**1.6E-01**	***2.6E+00***	NR	***2.7E+00***	***YES***
Mercury	NA	NA	NA	NA	NR	0.0E+00	NO
Molybdenum	4.0E-03	3.5E-04	4.3E-03	NA	NR	4.3E-03	NO
Nickel	1.5E-03	1.3E-02	1.5E-02	3.5E-02	NR	5.0E-02	NO
Selenium	2.6E-04	2.3E-05	2.8E-04	6.7E-06	NR	2.9E-04	NO
Silver	NA	NA	NA	NA	NR	NA	NO
Thallium	NA	NA	NA	NA	NR	NA	NO
Vanadium	2.1E-02	7.2E-02	9.4E-02	NA	NR	9.4E-02	NO
Zinc	2.5E-03	2.2E-04	2.8E-03	NA	NR	2.8E-03	NO
TOTAL gastrointestinal irritation (copper and iron)	5.5E-02	1.1E-02	6.6E-02	NR	NR	6.8E-02	NO
TOTAL neurotoxicity (aluminum, arsenic, cobalt, manganese, mercury, selenium)	**2.3E-01**	**1.5E-01**	**3.8E-01**	***3.6E+00***	NR	***4.0E+00***	***YES (aluminum, arsenic, cobalt, manganese)***
TOTAL nephrotoxicity (barium, cadmium, molybdenum, vanadium)	2.9E-02	7.6E-02	**1.1E-01**	9.6E-02	NR	**2.0E-01**	NO
TOTAL cardiovascular effects (arsenic, selenium)	8.1E-02	2.8E-02	**1.1E-01**	**1.7E-01**	NR	**2.8E-01**	NO

NA: lack of relevant COI data (either not detected or not analyzed) or missing toxicity values. NR: not relevant. **Bold** values exceed HQ of 0.1, and ***bold/italic*** values exceed HQ of 1.0. Only COIs with HQs exceeding 0.1 are summed, and then only if toxicologically relevant.
1: Retained on the basis of potential additive effects.

5.3.4.3 *Interpretation of Hazard Quotient Results*

The defined criteria for COIs carried forward for further evaluation as COPCs in Tier II, as previously stated, are exceeding a HQ of 1.0, or a particular COI with a HQ of between 0.1 and 1.0 in combination with other COIs with similar toxic effects exceeding a total HI of 1.0. These HQs and HIs are based upon maximum COI concentrations, as previously discussed. Note that these results are for screening only and should not be used for informing risk management decisions.

The results for Scenarios A, B, and D indicate that no COIs exceed the defined criteria for further evaluation in Tier II. Therefore, these scenarios are not evaluated further for non-carcinogenic COIs.

A number of COIs exceed the defined criteria in Scenario C (Table 11) for children, and thus are defined as COPCs. These include:

- Aluminum;
- Arsenic;
- Cobalt; and,
- Manganese.

Of these, aluminum is retained even though it has a HQ that is less than 1.0, because the HQ exceeds 0.1, and aluminum has similar toxic effects to other COIs that exceed HQs of 1.0. Note that the soil ingestion pathway dominates, with only manganese exceeding a HQ of 1.0 for dust inhalation.

In Scenario E (Table 13), the same COIs are retained as COPCs. All except manganese are retained because of similar toxic effects. Inhalation of dust is a major pathway.

In summary, aluminum, arsenic, cobalt, and manganese; and Scenarios C (residence) and E (construction), are retained for Tier II. Note that these are future scenarios. No current scenarios for non-carcinogenicity are carried forward.

5.3.4.4 *Incremental Lifetime Cancer Risk Results*

Results are presented in Table 14, Table 15, Table 16, Table 17, and Table 18 as individual COI ILCRs, plus ILCRs are combined as appropriate in terms of toxic effects. As previously discussed, the only COIs that have similar effects (i.e., lung cancer) are the carcinogenic COIs that have inhalation URs. In all cases, "NA" in tables refers to a lack of relevant COI data (either not detected or not analyzed) or missing toxicity values; thus no ILCR is estimated. "NR" means "not relevant".

ILCR values are presented as one significant figure per EPA guidance [EPA, 1989]. **Bold** values exceed an ILCR of 1E-06, and ***bold/italic*** values exceed an ILCR of 1E-05. Only individual COIs with ILCRs greater than 1E-05, or combinations of COIs with individual inhalation ILCRs greater than 1E-06 that exceed 1E-05 are carried forward into Tier II. Note that these results are for screening only and should not be used for informing risk management decisions. Interpretation of results follows the tables.

Table 14: Tier I Scenario A (Commercial Ranching) Incremental Lifetime Cancer Risks

COI	Soil Ingestion	Dermal Absorption	Soil Ingestion + Dermal Absorption	Dust Inhalation	Surface Water Ingestion	*Total*	Retain?
Arsenic	***1E-05***	**4E-06**	***2E-05***	6E-08	NA	***2E-05***	***YES***
Beryllium	NA	NA	NA	2E-09	NA	2E-09	NO
Cadmium	NA	NA	NA	2E-09	NA	2E-09	NO
Chromium VI	**2E-06**	***2E-05***	***2E-05***	6E-07	7E-07	***2E-05***	***YES***
Cobalt	NA	NA	NA	1E-07	NA	1E-07	NO
Nickel	NA	NA	NA	5E-09	NA	5E-09	NO
TOTAL lung cancer	NR	NR	NR	8E-07	NR	8E-07	NO

NA: lack of relevant COI data (either not detected or not analyzed) or missing toxicity values. NR: not relevant. **Bold** values exceed ILCR of 1E-06, and ***bold/italic*** values exceed ILCR of 1E-05.

Table 15: Tier I Scenario B (Trespassing) Incremental Lifetime Cancer Risks

COI	Soil Ingestion	Dermal Absorption	Soil Ingestion + Dermal Absorption	Dust Inhalation	Surface Water Ingestion	*Total*	Retain?
Arsenic	**3E-06**	9E-07	**4E-06**	7E-09	NA	**4E-06**	NO
Beryllium	NA	NA	NA	2E-10	NA	2E-10	NO
Cadmium	NA	NA	NA	2E-10	NA	2E-10	NO
Chromium VI	5E-07	**4E-06**	**5E-06**	8E-08	2E-07	**5E-06**	NO
Cobalt	NA	NA	NA	1E-08	NA	1E-08	NO
Nickel	NA	NA	NA	6E-10	NA	6E-10	NO
TOTAL lung cancer	NR	NR	NR	1E-07	NR	1E-07	NO

NA: lack of relevant COI data (either not detected or not analyzed) or missing toxicity values. NR: not relevant. **Bold** values exceed ILCR of 1E-06, and ***bold/italic*** values exceed ILCR of 1E-05.

Table 16: Tier I Scenario C (Residence) Incremental Lifetime Cancer Risks

COI	Soil Ingestion	Dermal Absorption	Soil Ingestion + Dermal Absorption	Dust Inhalation	Surface Water Ingestion	*Total*	Retain?
Arsenic	***6E-05***	**8E-06**	***7E-05***	2E-07	NR	***7E-05***	***YES***
Beryllium	NA	NA	NA	7E-09	NR	7E-09	NO
Cadmium	NA	NA	NA	7E-09	NR	7E-09	NO
Chromium VI	***2E-05***	***9E-05***	***1E-04***	**6E-06**	NR	***1E-04***	***YES***
Cobalt	NA	NA	NA	4E-07	NR	4E-07	NO
Nickel	NA	NA	NA	2E-08	NR	2E-08	NO
TOTAL lung cancer	NR	NR	NR	**7E-06**	NR	**7E-06**	NO

NA: lack of relevant COI data (either not detected or not analyzed) or missing toxicity values. NR: not relevant. **Bold** values exceed ILCR of 1E-06, and ***bold/italic*** values exceed ILCR of 1E-05.

Table 17: Tier I Scenario D (Recreation) Incremental Lifetime Cancer Risks

COI	Soil Ingestion	Dermal Absorption	Soil Ingestion + Dermal Absorption	Dust Inhalation	Surface Water Ingestion	*Total*	Retain?
Arsenic	**2E-06**	6E-07	**2E-06**	1E-08	NA	**2E-06**	NO
Beryllium	NA	NA	NA	4E-10	NA	4E-10	NO
Cadmium	NA	NA	NA	3E-10	NA	3E-10	NO
Chromium VI	4E-07	**4E-06**	**4E-06**	1E-07	2E-07	**5E-06**	NO
Cobalt	NA	NA	NA	3E-08	NA	3E-08	NO
Nickel	NA	NA	NA	8E-10	NA	8E-10	NO
TOTAL lung cancer	NR	NR	NR	2E-07	NR	2E-07	NO

NA: lack of relevant COI data (either not detected or not analyzed) or missing toxicity values. NR: not relevant. **Bold** values exceed ILCR of 1E-06, and ***bold/italic*** values exceed ILCR of 1E-05.

Table 18: Tier I Scenario E (Construction) Incremental Lifetime Cancer Risks

COI	Soil Ingestion	Dermal Absorption	Soil Ingestion + Dermal Absorption	Dust Inhalation	Surface Water Ingestion	*Total*	Retain?
Arsenic	5E-07	2E-07	6E-07	1E-07	NR	8E-07	NO
Beryllium	NA	NA	NA	4E-09	NR	4E-09	NO
Cadmium	NA	NA	NA	4E-09	NR	4E-09	NO
Chromium VI	8E-08	78E-07	8E-07	**1E-06**	NR	**2E-06**	NO
Cobalt	NA	NA	NA	3E-07	NR	3E-07	NO
Nickel	NA	NA	NA	1E-08	NR	1E-08	NO
TOTAL lung cancer	NR	NR	NR	**2E-06**	NR	**2E-06**	NO

NA: lack of relevant COI data (either not detected or not analyzed) or missing toxicity values. NR: not relevant. **Bold** values exceed ILCR of 1E-06, and ***bold/italic*** values exceed ILCR of 1E-05.

5.3.4.5 Interpretation of Incremental Lifetime Cancer Risk Results

The defined criteria for COIs carried forward for further evaluation as COPCs in Tier II, as previously stated, are individual COIs with ILCRs greater than 1E-05, or combinations of COIs with individual inhalation ILCRs greater than 1E-06 that exceed 1E-05. These ILCRs are based upon maximum COI concentrations, as previously discussed.

In all cases, the only COIs that exceeded the 1E-05 ILCR were arsenic and CrVI. In no cases did individual or summed inhalation ILCRs exceed this level. Arsenic and/or CrVI ingestion pathways exceeded a 1E-05 ILCR in Scenarios A (ranching) and C (residence). Thus, the COIs that will be carried forward as COPCs in these scenarios on the basis of carcinogenicity include the following:

- Arsenic
- Chromium VI

5.4 Tier I Screening Human Health Risk Assessment Summary

A Tier I screening was conducted for the LIU HHRA using maximum COI concentrations with conservative scenarios and exposure assumptions. The intent of Tier I was to identify the most risk-relevant scenarios and exposure pathways and to identify COPCs.

The following scenarios are carried forward into Tier II for non-carcinogenic COPCs:

- C: Residence
- E: Construction

These are future scenarios. Unacceptable risks from non-carcinogenic COPCs are not expected to exist for present-day scenarios. Soil ingestion, dermal absorption, and dust inhalation are all retained.

The following scenarios are carried forward into Tier II for carcinogenic COPCs:

- A: Ranching
- C: Residence

Inhalation ILCRs are not considered to be unacceptable in any scenario.

The only exposure pathway that can be consistently excluded from all of the scenarios, from both HQ and ILCR perspectives, is surface water ingestion. Therefore, surface water ingestion is not carried forward into Tier II.

The following COIs are therefore carried forward into Tier II as COPCs:

- Aluminum
- Arsenic
- Chromium VI
- Cobalt
- Manganese

Arsenic is the only COPC to be evaluated as both a carcinogen (oral and dermal pathways only) and a non-carcinogen. Table 19 is a summary of scenarios, exposure pathways, and COPCs carried forward into Tier II.

Table 19: Summary of Tier II Scenarios, Exposure Pathways, and Constituents of Potential Concern

Noncarcinogenic COPCS	Scenarios	Exposure Pathways (as appropriate for scenarios)	Carcinogenic COPCS	Scenarios	Exposure Pathways (as appropriate for scenarios)
Aluminum Arsenic Cobalt Manganese	C: Residence E: Construction	Soil Ingestion Dust Inhalation Dermal Absorption	Arsenic Chromium VI	A: Ranching C: Residence	Soil Ingestion Dermal Absorption

6 Tier II Human Health Risk Assessment

6.1 Introduction

The Tier II HHRA conducted here follows the same basic process as Tier I. The equations (Appendix I) are unchanged. The main differences include the following:

- A more limited set of exposure scenarios
- A more limited set of constituents (as COPCs)
- Assessment of reference area COPC concentrations
- Use of 95% upper confidence limits (UCLs) on COPC mean concentrations for EPCs
- More realistic exposure assumptions in some cases
- More realistic bioavailability fractions where possible
- More detail is provided regarding potential toxicity of COPCs at the concentrations present in order to provide informative context

6.2 Exposure Assessment

6.2.1 Estimation and Use of Exposure Point Concentrations

The Tier I analysis employed in the HHRA used maximum detected concentrations of all COIs for EPCs in exposure models detailed in Appendix I. Tier II uses 95% UCLs for COPC EPCs. The same requirements applied in Table 2 are applied here. As previously indicated, surface water was screened out by Tier I and is not evaluated in Tier II.

The analytical chemistry results for the COPCs (aluminum, arsenic, chromium, cobalt, manganese) from soil and sediment samples collected at the LIU site and reference locations were compiled in a data set. The reference locations include those collected for the LIU RI [Arcadis, 2012], and a second set of samples from reference locations collected for the Background Report [Chino, 1995] and used in the STSIU HHRA [Gradient, 2008]. Samples collected for the site-wide ecological RA, called the ERA reference data here, were also evaluated [Newfields, 2005]. The LIU site and LIU reference data include the results presented in the LIU RI report [Arcadis, 2012] that reflect current conditions. All sediment data from Tributary 2 superseded by the 2007-

2008 removal action were excluded. Data identified as field or laboratory duplicates were excluded.

Summary statistics for site and reference area data are presented in Appendix II. The data are summarized in separate groups representing the LIU site, LIU reference locations, and the STSIU/ERA reference locations. The tables in Appendix II summarize the overall number of samples; the number of detected concentrations (detects); the minimum, maximum, median (50th percentile), and arithmetic mean of detects; the number of nondetects (data qualified as "U"; i.e., analyte not detected above detection limit); and the minimum, maximum and arithmetic mean of the reported values for nondetects.

An EPC for Tier II represents a conservative estimate of the average concentration of a COPC in an environmental medium that a receptor would contact over time. The data sets used to calculate EPCs for each of the sample sets are described above. Consistent with EPA guidance [EPA, 1992a], data without qualifiers and data qualified as "U" (analyte not detected above detection limit) or "J" (analyte was positively identified, but concentration was estimated), were included in the EPC calculations. The Kaplan-Meier method or a substitution method using a value equal to one-half the reported detection limit was used to calculate EPCs for COPC concentrations reported as "U" or "non-detect", depending upon the number of nondetects and the magnitude of the detection limits.

EPA considers the arithmetic average or mean concentration of a COPC to be a reasonable estimate of the average concentration that is contacted over time at a site [EPA, 1989]. However, because of limitations and uncertainties inherent in all soil sampling plans, it is not possible to know the true mean concentration of a COPC at a site. Therefore, the 95% UCL on the mean concentration is used as a conservative estimate of the average exposure concentration [EPA, 1992b]. The 95% UCL equals or exceeds the true mean 95% of the time, and is appropriate to use when it is assumed that an individual has an equal probability of contacting any location within the exposure area. EPC concentrations were calculated according to current US EPA guidance and methods [EPA, 2010a]. For COPCs with few data points and considerable variability among the data points, the 95% UCL can be greater than the maximum concentration detected at the site. In these instances (here, only LIU reference sediment data), the maximum concentration was used as the EPC.

Only the arsenic data included a number of nondetects. Addressing nondetects in a statistical analysis involves professional judgment, as there is no consensus as to addressing concentrations of analytes that may or may not be 'real'. In the case of arsenic, two sets of means and UCLs are provided for comparative purposes; one calculated using the commonly applied Kaplan-Meier method, and the other calculated using ½ the analytical detection limit in lieu of zeros for nondetects.

The selected methods for estimating 95% UCLs involved the Student's t and BCA (bias-corrected and accelerated) bootstrap [EPA, 2010a;Efron, B. and Tibshirani, R., 1993] UCLs. The BCA bootstrap is the recommended bootstrap method to address nonparametric data [Efron, B. and Tibshirani, R., 1993]. For data sets that are normally (Gaussian) distributed (i.e. are not statistically significantly different from normal), a

95% Student's t UCL was selected. The 95% Student's t UCL was also selected for data sets with fewer than seven samples, as normality tests do not perfom well for such data sets, and bootstrap methods are not recommended. For larger data sets that are not normally distributed, the larger of the 95% Student's t UCL and the 95% BCA bootstrap UCL was chosen.

Table 20 below presents 95% UCLs and means for LIU site concentrations, and Table 21 and Table 22 present similar information for the two reference areas.

Table 20: Tier II Exposure Point Concentrations for LIU Site

COPC	Surface Soil (mg/kg 0-1"); 95%UCL	Surface Soil (mg/kg 0-1"); Mean	Reference	Distribution /Method	Shallow Soil (mg/kg 0-6"); 95% UCL	Shallow Soil (mg/kg 0-6"); Mean	Reference	Distribution /Method	Sediment (mg/kg 0-6"); 95% UCL	Sediment (mg/kg 0-6"); Mean	Reference	Distribution/ Method
Aluminum	17673	15451	[Chino, 1995; SRK, 2008; Arcadis, 2012]	Gamma/ BCA bootstrap	19191	16793	[Arcadis, 2012]	Normal/ Student's-t	10740	9809	[Chino, 1995; Arcadis JSA, 2001; Golder 2008; SRK, 2008; Golder, 2010; Arcadis 2012]	Gamma/BCA Bootstrap
Arsenic	7.6	5.1		Lognormal/ BCA bootstrap	9.8	5.58		Gamma/ BCA bootstrap	4.9 (5.8)[1]	4.1 (5.0)[1]		Gamma/ Kaplan-Meier BCA, (detection limit/2)
Chromium	31	22		Non-parametric/ BCA bootstrap	27	21		Gamma/ BCA bootstrap	25	19		Non-parametric/ BCA Bootstrap
Cobalt	12	11		Gamma/ BCA bootstrap	9.8	9.2		Normal/ Student's-t	12	11		Non-parametric/ BCA Bootstrap
Manganese	686	587		Normal/ Student's-t	637	574.8		Normal/ Student's-t	651	594		Non-parametric/ BCA Bootstrap

BCA: bias-corrected and accelerated
1: Values in parentheses estimated using the detection limit divided by 2 for nondetects.

Table 21: Tier II Exposure Point Concentrations for LIU Reference Area

COPC	Surface Soil (mg/kg 0-1"); 95%UCL	Surface Soil (mg/kg 0-1"); Mean	Reference	Distribution /Method	Shallow Soil (mg/kg 0-6"); 95% UCL	Shallow Soil (mg/kg 0-6"); Mean	Reference	Distribution/ Method	Sediment (mg/kg 0-6"); 95% UCL	Sediment (mg/kg 0-6"); Mean	Reference	Distribution/ Method
Aluminum	12280	10693	[Chino, 1995; Arcadis, 2012]	Normal/ Student's-t	12579	10548	[Arcadis, 2012]	Normal/ Student's-t	NA			
Arsenic	5.5	4.2		Normal/ Student's-t	6.8	4.3		Normal/ Student's-t				
Chromium	13	11		Normal/ Student's-t	18	12		Normal/ Student's-t				
Cobalt	16	13		Normal/ Student's-t	16	11		Normal/ Student's-t	6.0	5.1	[Chino, 1995]	Normal/used maximum
Manganese	972	757		Normal/ Student's-t	1171	737		Normal/ Student's-t	NA			

NA: not available or applicable

Table 22: Tier II Exposure Point Concentrations for STSIU/ERA Reference Areas

COPC	Surface Soil (mg/kg 0-1"); 95%UCL	Surface Soil (mg/kg 0-1"); Mean	Reference	Distribution/ Method	Shallow Soil (mg/kg 0-6"); 95% UCL	Shallow Soil (mg/kg 0-6"); Mean	Reference	Distribution /Method	Sediment (mg/kg 0-6"); 95% UCL	Sediment (mg/kg 0-6"); Mean	Reference	Distribution /Method
Aluminum	25395	22567	[Chino, 1995]	Normal/ Student's-t	57460	46442	[Arcadis, 2012]	Gamma/ Student's-t	NA			
Arsenic	2.7 (2.7)[1]	2.1 (2.1)[1]		Normal/ Student's-t (detection limit/2)	8.3	6.5		Normal/ Student's-t				
Chromium	44	36		Normal/ Student's-t	41	35		Normal/ Student's-t				
Cobalt	12	11		Normal/ Student's-t	18	14		Normal/ Student's-t				
Manganese	550	499		Normal/ Student's-t	1549	1291		Normal/ Student's-t				

NA: not available or applicable
1: Values in parentheses estimated using the detection limit divided by 2 for nondetects.

6.2.2 Reference Area Comparisons

To address the question of whether site concentrations are significantly greater than reference area concentrations, box plots and statistical hypothesis tests were employed [EPA, 2002].

Box plots of COPC concentrations provide a visual review of data sets and comparison of site concentrations to reference concentrations. These plots summarize information about the shape and spread of the distribution of concentrations from a data set. Box plots consist of a "box", a median line across the box, whiskers (lines extended beyond the box and terminated with a perpendicular line segment or whisker), and 'outside' points beyond the whiskers. The area enclosed by the box shows the concentration range containing the middle half of the data; that is, the lower box edge is at the first or lower quartile (i.e., 25th percentile), and the upper box edge is at the third or upper quartile of the data (i.e., the 75th percentile). The height of the box (the interquartile range) is a measure of the spread of the concentrations. The horizontal line across the box represents the median (50th percentile, or second quartile) of the data, which is a measure of the center of the concentration distribution. If the median line divides the box into two approximately equal parts, this indicates that the shape of the distribution of concentrations is approximately symmetric; if not, it indicates that the distribution is skewed or nonsymmetric. The set of concentrations are plotted as points overlaying the box plot with plotting characters, using "x" for detected concentrations and "o" for nondetects (only relevant for arsenic).

Figures 2 though 6 represent box plots for site and reference concentrations for each of the COPCs.

Figure 2: Comparisons of Site and Reference Concentrations for Aluminum

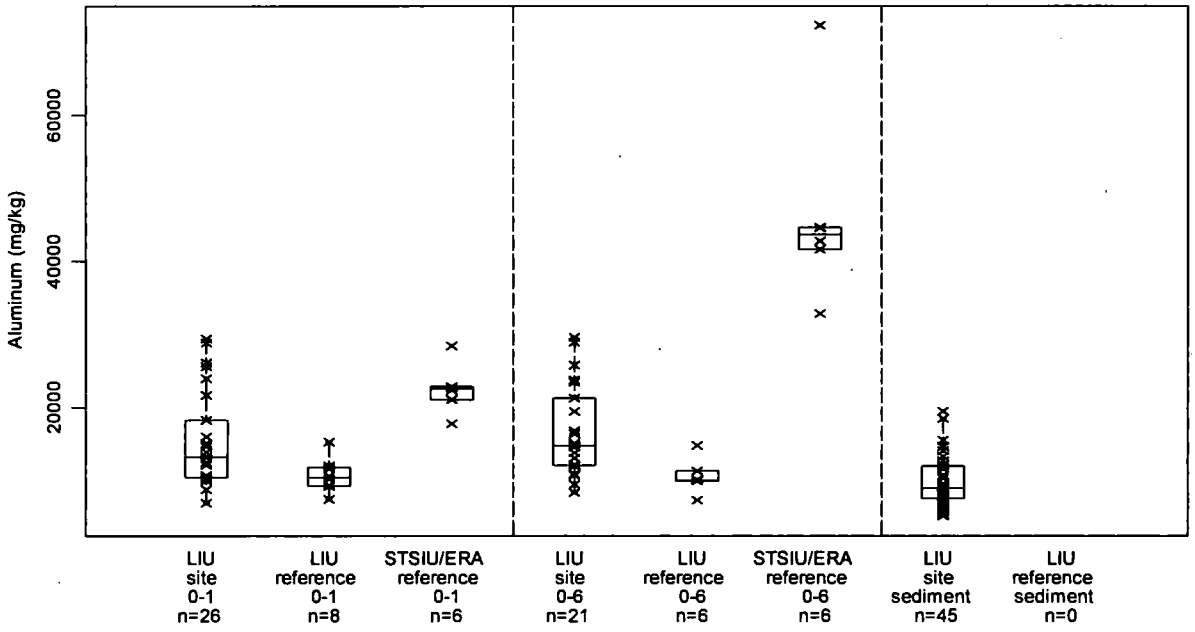


Figure 3: Comparisons of Site and Reference Concentrations for Arsenic

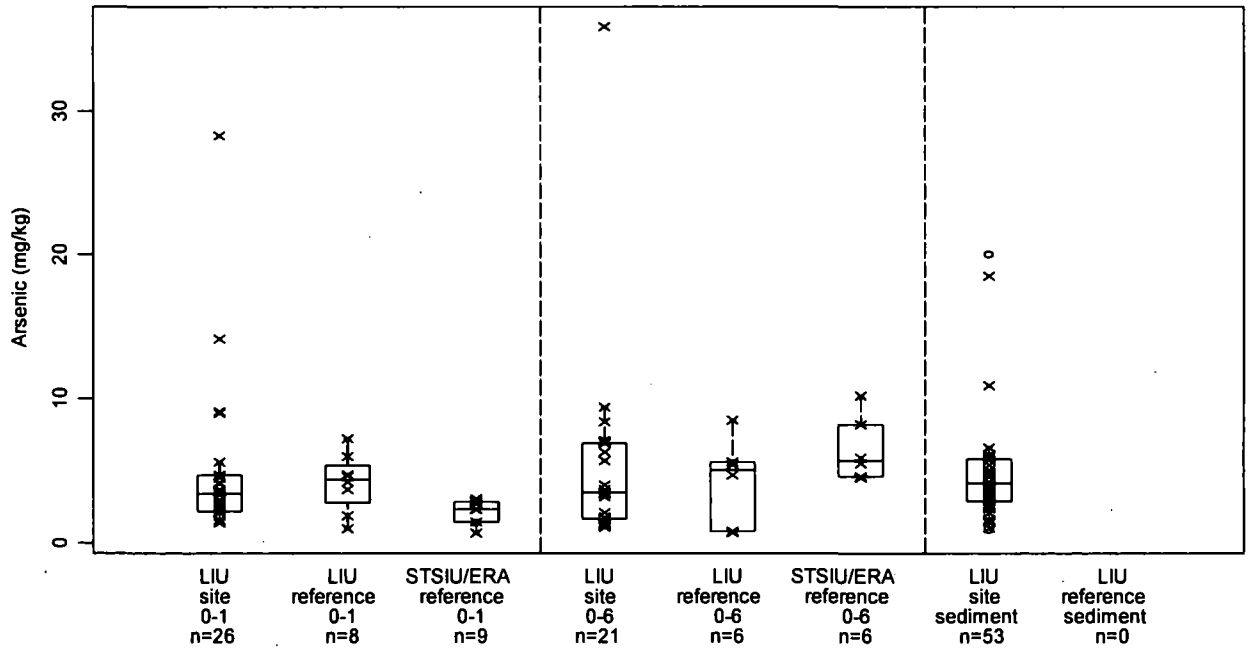


Figure 4: Comparisons of Site and Reference Concentrations for Chromium

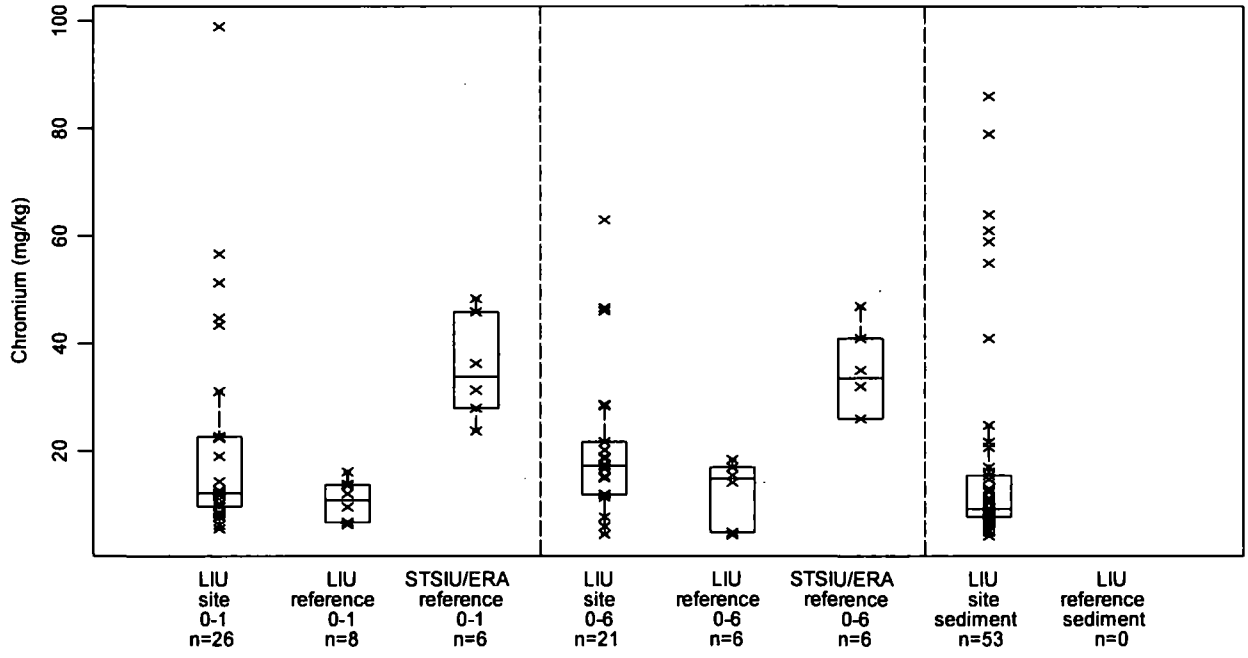


Figure 5: Comparisons of Site and Reference Concentrations for Cobalt

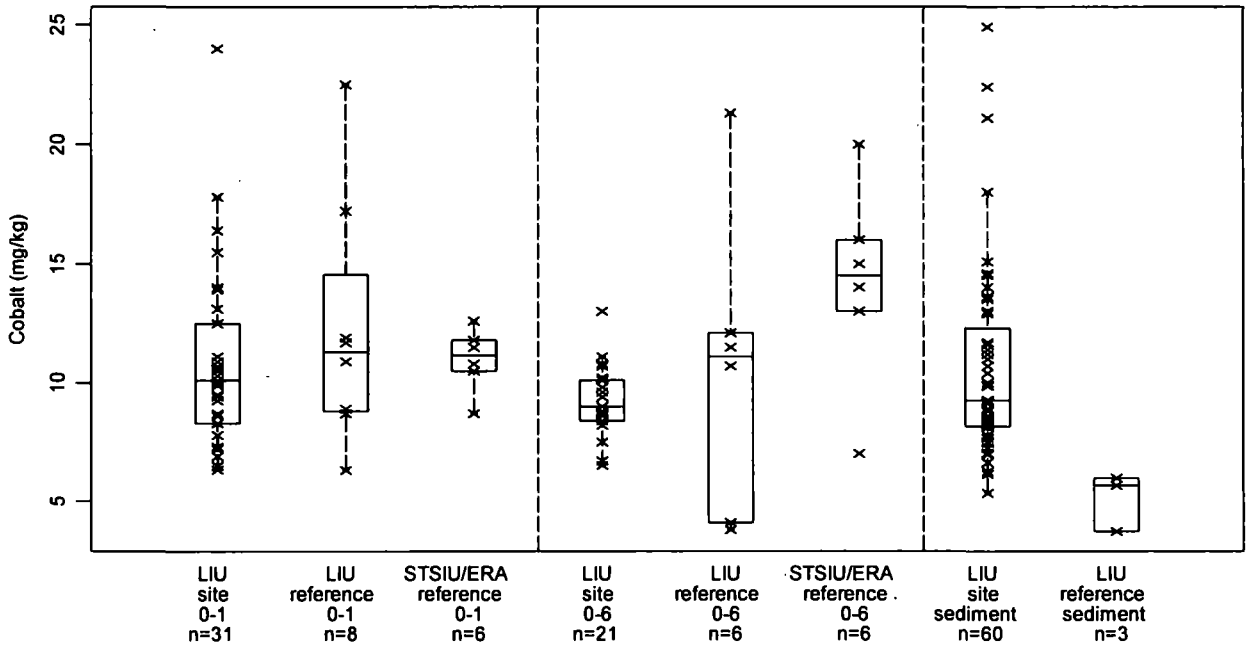
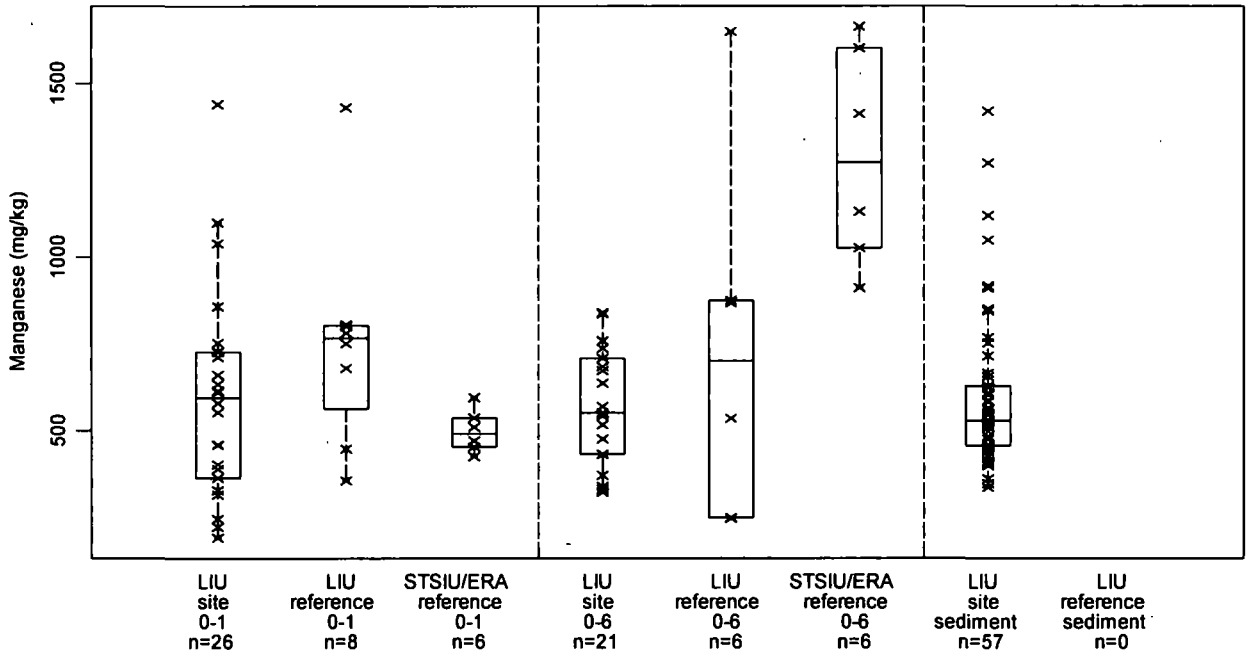


Figure 6: Comparisons of Site and Reference Concentrations for Manganese



Comparisons of site concentrations versus reference area concentrations do not identify a COPC that was consistently elevated for site samples. In fact, many of the STSIU/ERA reference area concentrations are higher than either LIU site or LIU reference concentrations. Likewise, sediment concentrations appear to be similar to soil concentrations.

Statistical tests provide greater detail with regard to whether the combined set of site concentrations is systematically greater than the reference set concentrations. Three tests are employed here. The Gehan test is the nonparametric (distribution-independent) equivalent of the familiar t-test and is designed to accommodate nondetects reported at multiple detection limits using a robust ranking method. It is identical to the Wilcoxon rank sum test when applied to results containing no nondetects. There are two tests designed to detect a shift or difference in the largest concentrations, or upper tail, of the sample distribution. The quantile test is used to determine whether there are significantly more site samples than reference samples in an upper percentile of the combined (site and reference) data results. The slippage test is used to determine whether there is a significant number of site samples larger than the maximum detected reference concentration.

The conclusion of a statistical reference area comparison test is indicated by the probability value or *p*-value associated with the test statistic. The *p*-value indicates the probability that the test statistic would be as-large or larger than the one calculated from the data, by chance alone, if the site and reference data came from the same distribution. It is standard practice to conclude that tests with *p*-values of less than 0.05 (or a less than one out of 20 chance) indicate a statistically significant difference between the sets of site and reference results.

The reference area comparisons were performed separately for surface soil samples and for shallow soil samples. Separate comparisons were performed comparing LIU site samples to the two sets of reference locations; i.e., the LIU RI reference locations and the STSIU and ERA reference locations. The LIU sediment samples did not have a reference set with an adequate number of samples to perform reference comparisons. Table 23 summarizes comparisons of LIU site samples with reference samples.

Table 23: Comparisons of Site and Reference COPC Concentrations

COPC	Site Set		Reference Set		*p*-Values of Statistical Tests			Conclusion
	Samples	*Detects*	*Samples*	*Detects*	*Gehan*	*Quantile*	*Slippage*	*Site Elevated?*
Surface Soil (0-1 Inch): LIU Site vs. LIU Reference								
Aluminum	26	26	8	8	**0.01**	0.13	0.09	**Yes**
Arsenic	26	26	8	8	0.74	0.92	0.24	No
Chromium	26	26	8	8	0.10	0.40	0.06	No
Cobalt	31	31	8	8	0.78	0.94	0.79	No
Manganese	26	26	8	8	0.95	0.99	0.76	No
Surface Soil (0-1 Inch): LIU Site vs. STSIU/ERA Reference								
Aluminum	26	26	6	6	0.99	1.00	0.66	No
Arsenic	26	26	9	8	**0.02**	**0.01**	**0.00**	**Yes**
Chromium	26	26	6	6	0.99	1.00	0.52	No
Cobalt	31	31	6	6	0.84	0.93	0.26	No
Manganese	26	26	6	6	0.26	0.06	**0.03**	**Yes**
Shallow Soil (0-6 Inch): LIU Site vs. LIU Reference								
Aluminum	21	21	6	6	**0.01**	0.06	**0.04**	**Yes**
Arsenic	21	21	6	6	0.40	0.32	0.60	No
Chromium	21	21	6	6	0.09	0.06	0.06	No
Cobalt	21	21	6	6	0.82	0.99	1.00	No
Manganese	21	21	6	6	0.69	0.93	1.00	No
Shallow Soil (0-6 Inch): LIU Site vs. STSIU/ERA Reference								
Aluminum	21	21	6	6	1.00	1.00	1.00	No
Arsenic	21	21	6	6	0.95	0.70	0.78	No
Chromium	21	21	6	6	0.99	0.99	0.78	No
Cobalt	21	21	6	6	0.99	1.00	1.00	No
Manganese	21	21	6	6	1.00	1.00	1.00	No

Bold values represent statistically significant results at the 0.05 level.

The reference area comparison tests did not identify a COPC that was consistently elevated for LIU site samples when considering comparisons to both LIU reference samples and other reference samples. This applied for both surface soil samples and shallow soil samples. Arsenic and manganese were identified as elevated in LIU site surface samples compared to STSIU/ERA reference samples. Aluminum was identified as elevated in LIU site surface samples compared to LIU reference samples. Aluminum was identified as elevated in LIU site shallow soil samples compared to LIU reference samples. Note that chromium is subject to uncertainty with regard to the proportion of CrVI versus CrIII, and this proportion might change from site to reference areas; therefore although total chromium concentrations do not appear to be elevated above reference areas, this may not reflect CrVI comparisons.

There were insufficient data to conduct statistical tests for differences between site and reference area sediment samples. However, examination of the box plots reveals that substantial differences do not appear to exist between site soils and site sediments.

Therefore, the conclusions above with regard to site soils versus reference area soils based upon the box plots may apply; but cannot be confirmed without more/better data.

6.2.3 Exposure Variable Assumptions

6.2.3.1 Introduction

The Tier I HHRA for the LIU included largely conservative values for the sake of 'protective' screening of COPCs. Some of these assumptions are modified for Tier II, as noted below, in order to reflect site-specific conditions, greater accuracy with regard to COPC characteristics, further literature research, or other considerations as noted. Assumptions that are unchanged are indicated by the phrase "*the same assumption applied in Tier I is applied here*". In all cases, the RME concept still applies and estimates of hazard and ILCR are protective of human health.

Tier II exposure variable values are presented in Table 24, along with references. Explanations are presented below. As in Tier I, note that some of the assumptions may vary from previous HHRAs. This is largely due to recent updated information in the latest EPA Exposure Factors Handbook [EPA, 2011].

Variables are presented in approximate alphabetical order below.

6.2.3.2 Averaging Time

The same assumption applied in Tier I is applied here. AT (in yr) is employed in the case of carcinogens to 'average' exposure over a lifetime, as there is assumed to be a lifetime risk of cancer upon exposure. A value of 78 yr is used for all scenarios. According to EPA [EPA, 2011], this is the mean (i.e., arithmetic mean, or average) lifespan (males plus females) as represented in (Table ES-1). For non-carcinogenic toxicants, the AT is equivalent to exposure duration (see below), as lifetime exposures are not assumed to be of interest in terms of toxicity (i.e., toxic effects do not persist throughout the lifetime of the receptor).

6.2.3.3 Bioavailability Fractions

Some of these assumptions have been changed. COPCs can absorb or bind to soil or dust particles, resulting in less ability for these compounds to be absorbed into the bloodstream. Relative bioavailability is the amount of a compound that is able to be absorbed into the bloodstream via soil related exposure routes, compared to the route of administration in the critical toxicity study; typically, soluble forms in water (or food; but additional adjustments must be made). Many metal compounds have low bioavailability in the gastrointestinal tract, and less via the skin.

For Tier II, the literature was examined as to whether values less-than 1.0 could be defensibly employed. It is beyond the scope of this HHRA to critically review this literature and the complexities of BFs in detail; rather, recent studies that employed defensible methodology are cited. However, note for example that estimates of BFs can be affected by factors such as speciation; and COPCs such as aluminum, iron, and manganese can affect absorption of other COPCs such as arsenic. It is possible that combinations of these COPCs could appreciably reduce bioavailability of any one COPC.

Addtionally, some studies measured bioaccessibility in *in vitro* studies that simulate passage through the gut (i.e., an acid environment in the stomach, and a basic environment in the intestines) versus bioavailability in *in vivo* studies. The latter were given preference here, if applicable. In most cases, standard extraction procedures (i.e., EPA digestion method 3050) were employed to characterize concentrations of COPCs in soils; however, in some cases the method was not provided. Thus, consideration of these and other uncertainties is important.

Findings for Tier II COPC ingestion BFs are summarized below.

- Aluminum: The bioaccessibility of alumimum and barium (see below) was measured in mining affected soils by Shock et al. [Shock, S. S., Bessinger, B. A., Lowney, Y. W., and Clark, J. L., 2007]. They found low bioaccessibility values of 0.0031 to 0.004 (i.e., 0.31 to 0.40 %). A RME estimate of 0.004 is used here.
- Arsenic: A number of *in vitro* and *in vivo* studies have examined arsenic, as it is often an important COPC at contaminated sites. The most recent studies include Roberts et al. [Roberts, S. M., Munson, J. W., Lowney, Y. W., and Ruby, M. V., 2007] and Bradham et al. [Bradham, K. D., Scheckel, K. G., Nelson, C. M., and et al., 2011]. These studies provide reviews of the previous literature as well. The Roberts et al. study used monkeys and a variety of soils, and found an average of approximately 0.17, with a maximum of 0.31 for an arsenic BF. The Bradham study used mice and soils from mining/smelting areas, and found an average of 0.33, with a maximum of 0.53. Based upon these studies and the likely conservatism of using mice versus monkeys (which have a similar GI tract to humans), a RME estimate of 0.31 (the maximum from the Roberts et al. study) is used here.
- Barium: The bioaccessibility of barium was measured in mining affected soils by Shock et al. [Shock, S. S., Bessinger, B. A., Lowney, Y. W., and Clark, J. L., 2007]. They found a wide range of bioaccessibility values of 0.0007 to 0.20. A RME estimate of 0.20 is used here.
- Chromium: Chromium as CrVI is of concern here. Bioavailability estimation is complicated by the different speciation of chromium in different soils. However, according to a review of the primary studies [Paustenbach, D. J., Bruce, G. M., and Chrostowski, P., 1997], the bioavailability of CrVI can range up to 0.08. A RME estimate of 0.08 is used here.
- Cobalt: No peer-reviewed published studies of cobalt bioavailability from soil were found. However, a recent large scale Canadian risk assessment study [SARA, 2008] conducted bioaccessibility studies for cobalt, and used a value of 0.28 for soil. A RME estimate of 0.28 is used here.
- Copper: The Hurley IU HHRA [Gradient, 2008] contracted with the University of Colorado to determine copper bioaccessibility in an *in vitro* study. This ranged from 0.48 to 0.78, with a mean of 0.65 and a 95% UCL of 0.69. This is within the range of other published studies. A RME estimate of 0.69 is used here.

- Iron: Sialelli et al. [Sialelli, J., Urquhart, G. J., Davidson, C. M., and Hursthouse, A. S., 2010] used an *in vitro* assay for urban soils, and found iron biaccessibilities from 0.10 to 0.18. A RME estimate of 0.18 is used here.
- Manganese: Sialelli et al. [Sialelli, J., Urquhart, G. J., Davidson, C. M., and Hursthouse, A. S., 2010] used an *in vitro* assay for urban soils, and found biaccessibilities from 0.12 to 0.41. A RME estimate of 0.41 is used here.
- Vanadium: The bioavailability of vanadium appears to be very low. Teng et al. [Teng, Y., Yang, J., Wang, J., and Song, L., 2011] found bioaccessibilities of 0.0001 to 0.041. A RME estimate of 0.041 is used here.

In Tier I, the values of BF_derm for most of the COPCs were taken from the Ontario Ministry of the Environment's soil standard development guidelines [Ontario MOE, 2011]. BF_derm may also be calculated as the absolute dermal absorption fraction (see Table 2.25 in [Ontario MOE, 2011], and Exhibit 3-4 in [EPA, 2004]) divided by the bioavailability of the COPC in the critical study related to the oral toxicity criterion ([EPA, 2004]; Exhibit 4-1). For the Tier I calculations, the larger of the calculated values for BF_derm and those published in the Ontario MOE guidance [Ontario MOE, 2011] was used. In a few cases these values exceeded 1.0; thus these BF values were set to 1.0.

For Tier II, these values were researched. In all cases aside from arsenic and CrVI, no additional or improved information was found; and thus the same information used in Tier I was employed.

For the following, BF_derm values were modified:

- Arsenic: Lowney et al. [Lowney, Y. W., Wester, R. C., Schoof, R. A., Cushing, C. A., Edwards, M., and Ruby, M. V., 2007] used monkeys to evaluate dermal biovailability of arsenic from various types of soils (wet and dry). The highest average value of 0.005 (wet Colorado soil) is used as a RME estimate here.
- Chromium: Chromium as CrVI is of concern here. Bioavailability estimation is complicated by the different speciation of chromium in different soils. However, Horowitz and Finley [Horowitz, S. B. and Finley, B. L., 1993] found a maximum of 0.001 CrVI bioaccessibility using a human sweat model. That value is used as a RME estimate here.

Table 25 is a summary of BFs.

6.2.3.4 Body Weight

The same assumption applied in Tier I is applied here. Exposures are adjusted by BW (in kg), as some other variables change depending upon the size of individual, and toxicity is typically body mass dependent.. According to EPA [EPA, 2011], a mean BW for adults (BW_a) over 21 is 80 kg (Table ES-1). For children (BW_c) from age 0 to 6 years in Scenario C, the time-weighted mean of the mean values for these age strata (15 kg) is used. For the first year, the time-weighted infant body weight is 7.8 kg. This value is then combined with body weights for ages 1 – <2 yr (11.4 kg), 2 – <3 yr (13.8 kg), and 3 – <6 yr (18.6 kg) for a time-weighted result of 14.8 kg, rounded to 15 kg.

6.2.3.5 *Dermal Surface Area*

The same assumptions applied in Tier I are applied here. The DSA (in cm^2) is an estimate of the exposed area of skin that would be available for contact with soil and dust (as much of the body is typically covered by clothes). Mean values are appropriate, as skin surface area is correlated with body weight. For adults, only the hands and arms are assumed to be exposed. The DSAs of arms and hands for adult males over 21 in EPA [EPA, 2011] (Table ES-1) are 3140 cm^2 and 1070 cm^2, respectively. For children, exposed skin is assumed to be available for arms, hands, legs, and feet. Skin surface area for a child age 3 – <6 yr is protectively used in the calculations. The mean DSAs from EPA [EPA, 2011] (Table ES-1) are; arms (1060 cm^2), hands (370 cm^2), legs (1950 cm^2), and feet (490 cm^2).

6.2.3.6 *Dermal Soil Adherence Factor*

The same assumptions applied in Tier I are applied here. The DSAF (in mg/cm^2) is a measure of how much soil is retained by the skin during particular activities. The assumption here is that exposure events (as represented in EPA [EPA, 2004].) are integrated over the period of a day, consistent with later EPA guidance [EPA, 2011] that no longer represents DSAF on a per-event basis. Note that it is possible to have a high degree of adherence when activities are conducted in mud or wet sediment; however, assuming that all activities will take place in such media is unrealistic.

For adults, the mean soil adherence for construction activities (the highest adherence) from EPA [EPA, 2011] (Table ES-1) for arms (0.1859 mg/cm^2) and hands (0.2763 mg/cm^2) is used. For children, mean soil adherence for "activities with soil" is used for arms (0.046 mg/cm^2), hands (0.17 mg/cm^2), legs (0.051 mg/cm^2), and feet (0.20 mg/cm^2).

6.2.3.7 *Exposure Duration*

The same assumptions applied in Tier I are applied here. RME EDs (in yr) are employed here, per EPA "standard default factors" [EPA, 1991]. For adults, an ED of 25 yr for Scenarios A and E, 30 yr is used for Scenarios B and D, and 24 yr (i.e., 30 minus 6 yrs for children) for Scenario C. For children, an ED of 6 yr is used to match the age range of concern in Scenario C.

6.2.3.8 *Exposure Frequency*

Some of these assumptions have been changed. RME EFs (in d/yr) represent the amount of time that receptors are expected to spend conducting activities in each of the scenarios. For the Tier I Scenario A EF, a value of 250 d/yr was applied. However, based upon information from the current leaseholder, ranching is conducted on a seasonal basis, thus this value may represent an upper bound. Therefore, this value is reduced for Tier II to a RME estimate of 180 d/yr, based upon the climate of the area and typical ranching practice. For Tier I Scenarios B and D, a professional judgment of 50 d/yr was assumed, based upon once per week, 50 wks per yr. However, this does not account for the low likelihood that the LIU would be the sole area in which trespassing or recreation would occur, thus this value may represent an upper bound. Therefore, this RME estimate is reduced to 25 d/yr for Tier II. For Scenario C, the standard default residential EF of 350

d/yr [EPA, 1991] is retained for both adults and children. The EF for Scenario E represents the EPA standard default values for a RME worker; i.e., 250 d/yr [EPA, 1991].

6.2.3.9 Exposure Time

The same assumptions applied in Tier I are applied here. RME ETs (in hr/d) represent the amount of time that receptors are expected to spend 'on site'. A typical working day is assumed to be 8 hrs, and defines ETs for adults in Scenarios A and E. It is also assumed that trespassers in Scenario B and recreationists in Scenario D would not spend more than 8 hr/d in the affected area. Thus, the same ET is protectively used for adults in these scenarios. For Scenario C, it is assumed that adults will spend 8 hr/d away from home, thus ET_a is 16 hr/d. This estimate is consistent with the sum of mean time spent indoors (Table 16-1; approximately 900 minutes) and outdoors (Table 16-22; approximately 140 minutes) at a residence [EPA, 2011]. Mean estimates of time indoors at a residence from birth to age 6 years vary between approximately 65% and 75% [EPA, 2011] (Table 16-1). For Tier II, it is protectively assumed that children in this scenario will spend 24 hr/d at home.

6.2.3.10 Fraction of Ingestion/Dermal Contact Associated with Site

The same assumptions applied in Tier I are applied here. For soil/dust ingestion and dermal exposure to soil, it is useful to include a FS factor (dimensionless) that accounts for exposure for some fraction of time off-site; i.e., to noncontaminated soil and dust. For the purpose of Tier II, however, this factor will be set to 1.0; thus assuming all soil contact is with site soils.

6.2.3.11 Ingestion Rate of Soil and Dust

The same assumptions applied in Tier I are applied here. Adults are assumed to ingest soil and dust at the same rate across scenarios. EPA [EPA, 2011] does not provide an upper-percentile value for adults. The value in Table 1 (100 mg/d) represents the "central tendency" estimate for ages 6 to 21, which is more conservative than the "adult" estimate (50 mg/d) in EPA [EPA, 2011] (Table ES-1). The value for children (200 mg/d) is an "upper percentile" estimate for children aged 3 to 6 [EPA, 2011] (Table ES-1).

Table 24: Tier II Exposure Assumptions

		A Ranching		*B Trespass*		*C Residence*		*D Recreation*		*E Construction*	
		Value	Reference	Value	Reference	Value	Reference	Value	Reference	Value	Reference
Variable	Units										
AT: carcinogens	yr	78	[EPA, 2011], Table ES-1	78	[EPA, 2011], Table ES-1	78	[EPA, 2011], Table ES-1	78	[EPA, 2011], Table ES-1	78	[EPA, 2011], Table ES-1
AT: noncarcinogens	yr	Equal to exposure duration	--	Equal to exposure duration	--	Equal to exposure duration	--	Equal to exposure duration	--	Equal to exposure duration	--
BW_a	kg	80	[EPA, 2011], Table ES-1	80	[EPA, 2011], Table ES-1	80	[EPA, 2011], Table ES-1	80	[EPA, 2011], Table ES-1	80	[EPA, 2011], Table ES-1
BW_c	kg	--	--	--	--	15	[EPA, 2011], Table ES-1	--	--	--	--
DSA_a	cm^2	3140; 1070	[EPA, 2011], Table ES-1 (see text)	3140; 1070	[EPA, 2011], Table ES-1 (see text)	3140; 1070	[EPA, 2011], Table ES-1 (see text)	3140; 1070	[EPA, 2011], Table ES-1 (see text)	3140; 1070	[EPA, 2011], Table ES-1 (see text)
DSAF_a	mg/cm^2	0.1859; 0.2763	[EPA, 2011], Table ES-1 (see text)	0.1859; 0.2763	[EPA, 2011], Table ES-1 (see text)	0.1859; 0.2763	[EPA, 2011], Table ES-1 (see text)	0.1859; 0.2763	[EPA, 2011], Table ES-1 (see text)	0.1859; 0.2763	[EPA, 2011], Table ES-1 (see text)
DSA_c	cm^2	1060; 370; 1950; 490	[EPA, 2011], Table ES-1 (see text)	1060; 370; 1950; 490	[EPA, 2011], Table ES-1 (see text)	1060; 370; 1950; 490	[EPA, 2011], Table ES-1 (see text)	1060; 370; 1950; 490	[EPA, 2011], Table ES-1 (see text)	1060; 370; 1950; 490	[EPA, 2011], Table ES-1 (see text)
DSAF_c	mg/cm^2	0.046; 0.17; 0.051; 0.2	[EPA, 2011], Table ES-1 (see text)	0.046; 0.17; 0.051; 0.2	[EPA, 2011], Table ES-1 (see text)	0.046; 0.17; 0.051; 0.2	[EPA, 2011], Table ES-1 (see text)	0.046; 0.17; 0.051; 0.2	[EPA, 2011], Table ES-1 (see text)	0.046; 0.17; 0.051; 0.2	[EPA, 2011], Table ES-1 (see text)
ED_a	yr	25	[EPA, 1991]	30	[EPA, 1991]	24	[EPA, 1991]	30	[EPA, 1991]	1	[EPA, 1991]
ED_c	yr	--	--	--	--	6	[EPA, 1991]	--	--	--	--
EF_a	d/yr	180	See text	25	See text	350	[EPA, 1991]	25	See text	250	[EPA, 1991]
EF_c	d/yr	--	--	--	--	350	[EPA, 1991]	--	--	--	--
ET_a	hr/d	8	See text	4	See text	16	See text	8	See text	8	See text
ET_c	hr/d	--	--	--	--	24	See text	--	--	--	--
IR_s_a	mg/d	100	[EPA, 2011], Table ES-1	100	[EPA, 2011], Table ES-1	100	[EPA, 2011], Table ES-1	100	[EPA, 2011], Table ES-1	100	[EPA, 2011], Table ES-1
IR_s_c	mg/d	--	--	--	--	200	[EPA, 2011], Table ES-1	--	--	--	--

--: not applicable

Table 25: Tier II Relative Bioavailability Factors

COPC	*Relative Dermal Bioavailability*	*Reference*	*Relative Oral Bioavailability*	*Reference*
Aluminum	0.01	Same as Tier I (see Table 5)	0.004	[Shock, S. S., Bessinger, B. A., Lowney, Y. W., and Clark, J. L., 2007]
Arsenic	0.005	[Lowney, Y. W., Wester, R. C., Schoof, R. A., Cushing, C. A., Edwards, M., and Ruby, M. V., 2007]	0.31	[Roberts, S. M., Munson, J. W., Lowney, Y. W., and Ruby, M. V., 2007]
Barium	0.14	Same as Tier I (see Table 5)	0.20	[Shock, S. S., Bessinger, B. A., Lowney, Y. W., and Clark, J. L., 2007]
Chromium VI	0.001	[Horowitz, S. B. and Finley, B. L., 1993]	0.08	[Paustenbach, D. J., Bruce, G. M., and Chrostowski, P., 1997]
Cobalt	0.01	Same as Tier I (see Table 5)	0.28	[SARA, 2008]
Copper	0.06	Same as Tier I (see Table 5)	0.69	[Gradient, 2008]
Iron	0.01	Same as Tier I (see Table 5)	0.18	[Sialelli, J., Urquhart, G. J., Davidson, C. M., and Hursthouse, A. S., 2010]
Manganese	0.25	Same as Tier I (see Table 5)	0.41	[Sialelli, J., Urquhart, G. J., Davidson, C. M., and Hursthouse, A. S., 2010]
Vanadium	0.38	Same as Tier I (see Table 5)	0.041	[Teng, Y., Yang, J., Wang, J., and Song, L., 2011]

All BFs are dimensionless.

6.3 Toxicity Assessment

6.3.1 Introduction

The bases for and assumptions inherent in EPA's chronic toxicity values (RfDs, RfCs, SFs, and URs) were discussed in Tier I. The literature bases for specific values were reviewed for Tier II. Although in many cases the toxicological basis for these values could be questioned and/or different studies employed to derive such values, such an effort would consume considerable time and resources. It is beyond the scope of this HHRA to conduct exhaustive toxicity reviews of the primary literature. Thus, the same EPA recommended values applied in Tier I for screening in IRIS [EPA, 2012a] or the RSL tables [EPA, 2012c] are applied for evaluating COPCs in Tier II. All values represent chronic exposures, as subchronic or acute exposures are not of interest in the HHRA.

All values are represented as RfDs, RfCs, or SFs, as opposed to alternative designations such as "provisional" RfDs. The reason this distinction is not made here is that regulatory HHRAs are generally constrained to use toxicity values published or recommended by EPA, regardless of the provisional or final nature of these values. The standard applied here is; if the RSL tables [EPA, 2012c] provide toxicity values, then they are employed here. No information is provided for potential carcinogenic inhalation URs for arsenic, CrVI, or cobalt; as these COPCs were screened out in Tier I for inhalation carcinogenicity.

An assumption here is that the "critical" toxic effects for chronic exposure identified by EPA or other regulatory agencies are the main effects of concern in the HHRA. These effects are the basis for the estimated toxicity values, and are generally considered to be the most relevant to public health. However, many of the COPCs can and do have multiple types of toxic effects in different tissues/organs (depending upon dose). For example, most metal compounds will irritate the lungs if inhaled. It is beyond the scope of this HHRA to account for all of the potential toxic effects of the COPCs; rather, the focus here is upon the critical effects identified by the agencies.

All of the COPCs are naturally-occurring, thus any populations in areas where these COPCs are prevalent will have some exposure via water and food. It would be impossible in most cases to separate out particular sources of exposure to these COPCs with any degree of precision or accuracy. Indeed, some of the COPCs are essential elements for human health. A brief discussion of these considerations follows the Toxicity Assessment.

All of the toxicity values are subject to uncertainty. EPA addresses some types of uncertainty by application of UFs to NOAELS or LOAELS, or by use of confidence bounds for SFs. Functionally, this practice adjusts toxicity values so that they are more conservative. However, there are many other sources of uncertainty, some of which are discussed in the Uncertainty Assessment section. For the purpose of the present section, it is worth noting that a major source of uncertainty exists with regard to the level of information readily available (i.e.; via the published literature and internet sources)

regarding the basis for many published toxicity values. For example, HEAST [EPA, 1997] contains limited information or discussion. In these cases, the discussion of the basis for toxicity values is necessarily brief. In several cases the original toxicology studies represented in all databases were published in reports or articles that could not be located or retrieved via online sources, often due to the advanced age of the publications (e.g., 1960s or earlier). A limitation of the use of published toxicity values is that this 'information source uncertainty' is not readily characterized. Additionally, the quality of older studies conducted prior to modern standards for laboratory and epidemiological research must be questioned.

Following are summaries of the toxicity of each of the COPCs, including qualitative confidence levels where provided. The main sources of information are the regulatory sources that estimated the toxicity values. Citations are generally only provided for primary studies or analyses upon which the toxicity values are based.

6.3.2 Aluminum

6.3.2.1 Introduction

Aluminum toxicity values are not listed in IRIS [EPA, 2012a]. The RSL tables [EPA, 2012c] list provisional values. The main source of the information below is the aluminum PPRTV documentation [EPA, 2006]. Please consult that report for further details and primary references.

Aluminum is the third most common element, and the most common metal in the Earth's crust (http://en.wikipedia.org/wiki/Abundance_of_elements_in_Earth's_crust). It is also a component of manufactured products in the building, automobile and container industries. Aluminum as a powder is a component in a number of consumer products, such as paints and fireworks. Aluminum complexes and minerals are used in the brewing and paper industries, and as coagulants for water purification. Aluminum oxide is a component of abrasives, catalysts, absorbents, and fillers. Aluminum chloride is a component of deodorants and anti-perspirants. Human exposure to aluminum largely occurs via food, water, food additives, packaging, cooking utensils, and medications (e.g.; antacids, buffered aspirin, anti-ulcer and anti-diarrheal formulations). Patients who routinely take aluminum-containing medications can receive much higher doses than would be experienced in a normal diet.

6.3.2.2 Non-Cancer Chronic Toxicity

The aluminum RfD and RfC are PPRTVs. There are no current values on IRIS [EPA, 2012a]. Although subject to peer-review, they have not gone through the extensive review that is associated with values in IRIS; and thus are classified as "provisional".

Oral Exposure

Aluminum's main toxic endpoints of interest are neurotoxicity and neurodevelopmental effects (i.e.; effects upon development of the nervous system in developing animals). There are no human data suitable for determination of an oral RfD; although there are suggestions that oral exposure can result in neurological effects. Neurobehavioral deficits have been observed in mice and rats exposed during various stages of development and

in subchronic studies. These deficits include impaired learning, changes in grip strength, altered startle response, and impaired motor coordination. In addition, several studies have shown that aluminum can produce changes in the central nervous system. Furthermore, aluminum has been shown to inhibit the gastrointestinal absorption of calcium, but it is not clear whether calcium deprivation enhances the neurotoxicity of aluminum, or whether aluminum exacerbates the adverse effects of calcium deprivation.

A LOAEL of 100 mg/kg-d for minimal neurotoxicity in the offspring of mice exposed to dietary aluminum lactate (soluble aluminum) during gestation and lactation is the basis for the RfD [Donald, J. M., Golub, M. S., Gershwin, M. E., and Keen, C. L., 1989;Golub, M. S., Han, B., Keen, C. L., Gershwin, M. E., and Tarara, R. P., 1995]. The LOAEL is considered minimal because the results of postweaning neurobehavioral tests indicate that performance deficits may be marginal, and effects did not persist after stopping exposure. Application of an uncertainty factor (UF) of 100 (3 for use of a minimal LOAEL, 10 for interspecies extrapolation and 3 for intrahuman variability where the critical effects have been observed in a sensitive sub-group) results in a provisional RfD of 1.0 mg/kg-d. This RfD is approximately 3-fold higher than the estimated normal daily aluminum intake of up to 3.0E-01 mg/kg-d (see the Non-Site Related Exposures to and Nutritional Essentiality of COPCs section below). There was low overall confidence in the RfD.

Inhalation Exposure

Aluminum has been determined as a likely cause for psychomotor and cognitive effects (particularly impaired coordination) via inhalation exposure in aluminum production workers and welders. The basis of the RfC is an occupational study [Hosovski E., Masticlica Z., Suderic D., and Radulovic D., 1990], in which workers were exposed to a time-weighted average (TWA) concentration of 4.6 to 11.5 mg/m^3 for an average of 12 years. Using 4.6 mg/m^3 as the LOAEL for psychomotor and cognitive impairment for an 8-hour occupational exposure, and corrections for discontinuous exposure (10 m^3/20 m^3 and 5 days/7 days), the adjusted LOAEL is 1.64 mg/m^3. Applying an uncertainty factor of 300 for intrahuman variability (10), use of a LOAEL (10) and an incomplete database (3) yields a provisional RfC of 5.0E-3 mg/m^3. There was low to medium confidence in the RfC.

6.3.2.3 Carcinogenicity

According to EPA [EPA, 2006], there is inadequate information to assess the carcinogenicity of aluminum via oral exposure.

6.3.3 Arsenic

6.3.3.1 Introduction

The main source of the information below is the arsenic IRIS documentation [EPA, 2012a] and supporting information [EPA, 2010b]; plus California EPA [CalEPA, 2008a;CalEPA, 2008b]. Please consult those sources for further details and primary references.

Arsenic is approximately the 50th most abundant element in the Earth's crust (http://en.wikipedia.org/wiki/Abundance_of_elements_in_Earth's_crust). Arsenic leaches from natural weathering of soil and rock into water, and low concentrations of arsenic are found in water, food, soil, and air. Industrial activities such as coal combustion and smelting operations can release higher concentrations of arsenic to the environment. Both inorganic and organic forms of arsenic exist; typically environmental concerns are associated with the more toxic inorganic forms. Arsenic is used for hardening copper and lead alloys. It also is used in glass manufacturing as a decolorizing and refining agent, as a component of electrical devices, in the semiconductor industry, and as a catalyst in the production of ethylene oxide. Arsenic compounds are used as a mordant in the textile industry, for preserving hides, as medicinals, pesticides, pigments, and wood preservatives. Production of chromate copper arsenate (a wood preservative) and arsenic containing pesticides accounts for about 90% of the domestic consumption of arsenic.

6.3.3.2 Non-Cancer Chronic Toxicity

IRIS [EPA, 2012a] lists an oral RfD for arsenic. There is no IRIS or PPRTV RfC; thus the California EPA value is used here, in accordance with the RSL tables [EPA, 2012c].

Oral Exposure

Arsenic can cause a variety of effects depending upon dose. The main endpoint of interest with regard to environmental doses of arsenic is a skin disease called blackfoot disease, which is characterized by blackened lesions and hardening of the skin on the bottom of the foot. A number of studies have been performed in human populations exposed to high naturally occurring arsenic. Tseng [Tseng, W. P., 1977] reported an increased incidence of blackfoot disease that increases with age and dose in a Taiwanese population. The low dose in this study (mean arsenic concentration of 170 μg/L) is considered a LOAEL. The control group (mean arsenic concentration of 9 μg/L) described in an earlier study [Tseng, W. P., Chu, H. M., How, S. W., Fong, J. M., Lin, C. S., and Yeh, S., 1968] showed no evidence of skin lesions. This group is considered a NOAEL. The NOAEL and LOAEL doses for both food and water estimated by EPA are as follows:

LOAEL: (170 μg/L x 4.5 L/d + 2 μg/d contribution of food) / 55 kg = 14 μg/kg-d

NOAEL: (9 μg/L x 4.5 L/d + 2 μg/d contribution of food) / 55 kg = 0.8 μg/kg-d.

The high water consumption rate and low body weight, compared to typical EPA defaults, were judgments on EPA's part regarding characteristics of this population. A number of other studies are cited in IRIS [EPA, 2012a] that support these toxicity values.

An UF of 3 was applied to the NOAEL to account for both the lack of data to preclude reproductive toxicity as a critical effect and to account for uncertainty as to whether the NOAEL of the critical study accounts for all sensitive individuals. Thus, the oral RfD is estimated as 3.0E-04 mg/kg-d. There was medium confidence in the RfD.

Inhalation Exposure

Due to the lack of an IRIS RfC, in accordance with the RSL tables [EPA, 2012c] a California EPA value is used here [CalEPA, 2008b;CalEPA, 2008a]. The California EPA

chronic Reference Exposure Level (REL) value for arsenic was specifically derived for neurological endpoints in children. The REL was extrapolated from a drinking water study. Please see Appendix D of [CalEPA, 2008a] for details.

The study of Wasserman et al. [Wasserman, G. A., Liu, X., Parvez, F., Ahsan, H., Factor-Litvak, P., van, Geen A., Slavkovich, V., LoIacono, N. J., Cheng, Z., Hussain, I., Momotaj, H., and Graziano, J. H., 2004] indicated a dose-response of decreasing Full-Scale intellectual function raw scores with increasing drinking water arsenic exposure in 10-year olds. The slope of the fitted quadratic model indicated a low dose slope of -0.44 points per µg/L. Assuming that an adverse effect level is a 1.0 point loss, then the corresponding arsenic concentration was calculated as:.

-1.0 point/-0.44 point per µg/L = 2.27 µg/L

California EPA states that this "might be"equivalent to a LOAEL. Assuming a drinking water intake based upon the 95% UCL for US children aged 1 to 10 years of 1.564 L/d the daily oral intake at this LOAEL was estimated at 3.6 µg/d. California EPA then converted this oral LOAEL to an inhalation LOAEL by assuming that 10-year old males would inhale 9.9 m^3/d. Assuming a lung absorption of 50 %, a value of 0.46 µg/m^3 was estimated. Applying a 3-fold UF for an estimated LOAEL based upon a quantitative dose response analysis and 10-fold for inter-individual variation (as only 10-year olds were studied), a health protective air concentration of 0.015 µg/m^3 was calculated. Thus, a value of 1.5E-05 mg/m^3 is used as a RfC in the RSL tables. A confidence level was not provided by California EPA. Note that this is the lowest value estimated by California EPA, and that detailed explanation of justification for the extrapolation from oral exposure to inhalation exposure in terms of toxic effects was not provided.

6.3.3.3 Carcinogenicity

IRIS [EPA, 2012a] lists an oral SF arsenic. Note that this value was last revised in 1998, and the primary studies are much older. EPA is currently in the process of reassessing arsenic carcinogenicity [EPA, 2010b].

A large amount of epidemiological evidence exists to support human carcinogenicity of arsenic via ingestion [EPA, 2010b]. Arsenic potentially causes both skin cancer and internal organ (liver, kidney, bladder) cancers, but the current SF focuses upon skin cancer as the critical endpoint of interest, as the greatest amount of information is available for this type of cancer. It is considered a Class A carcinogen based upon human evidence.

The same Tseng et al. studies used to develop the RfD [Tseng, W. P., 1977;Tseng, W. P., Chu, H. M., How, S. W., Fong, J. M., Lin, C. S., and Yeh, S., 1968] were used by EPA to develop a dose-response relationship for arsenic. Details of the assessment are found in previous versions of the EPA toxicological review of arsenic [EPA, 1988a;EPA, 1988b]. Briefly, the number of persons at risk over three dose intervals and four exposure durations, for males and females separately, were estimated from the reported skin cancer prevalence rates as percentages. It was assumed that the population had a constant exposure from birth, and that males consumed 3.5 L drinking water/d and females consumed 2.0 L/d. Doses were converted to equivalent doses for US males and females

based upon differences in body weights and differences in water consumption. It was assumed that skin cancer risk in the US population would be similar to the Taiwanese population (although this type of skin cancer is very rare in the US). A multistage model with time was used to predict dose-specific and age-specific skin cancer prevalance rates associated with ingestion of arsenic. Both linear and quadratic model fitting of the data were conducted. The maximum likelihood estimate of skin cancer risk for a 70 kg person drinking 2 L of water/d ranged from 1.0E-03 to 2.0E-03 for an arsenic intake of 1.0 μg/kg/d; the midpoint of which results in a SF of 1.5 per mg/kg-d.

This assessment was based upon prevalence of skin cancer rather than mortality, because the types of skin cancer studied are not normally fatal. However, competing mortality from blackfoot disease (see the RfD discussion above) would cause the risk of skin cancer to be underestimated. Other sources of inorganic arsenic such as food sources were not considered. There is also uncertainty associated with the assumed amount of water consumed per day by Taiwanese males, and the temporal variability of arsenic concentrations in specific wells was not known. As 'clean' tap water was supplied to many areas after 1966, the arsenic-containing wells were only used in dry periods. Because of the study design, particular wells used by those individuals developing skin cancer could not be identified, and arsenic intake could not be assigned except by village.

Eastern Research Group, under contract to EPA, convened an Expert Panel on Arsenic Carcinogenicity in 1997 (cited as Eastern Research Group 1997, in [EPA, 2012a]. Note that this report could not be located). According to the IRIS profile [EPA, 2012a], the Expert Panel stated; "it is clear from epidemiological studies that arsenic is a human carcinogen via the oral and inhalation routes (p. 20)." They also concluded, ". . . one important mode of action is unlikely to be operative for arsenic". The panel agreed that arsenic and its metabolites do not appear to directly interact with DNA (pp. 30-31)." In addition, the panel agreed that, "for each of the modes of action regarded as plausible, the dose-response would either show a threshold or would be nonlinear (p. 31)". The panel agreed, however, "that the dose-response for arsenic at low doses would likely be truly nonlinear, i.e., with a decreasing slope as the dose decreased. However, at very low doses such a curve might be linear but with a very shallow slope, probably indistinguishable from a threshold (p. 31)." The bases for these statements have not changed substantially since the time of this Panel. Despite much evidence that the MOA of arsenic skin carcinogenicity involves nonlinearity and/or a threshold, EPA applied the LNT hypothesis to the Tseng et al. data to derive the oral SF. Therefore, the SF is likely to be more conservative than if a threshold model had been applied.

A summary of the basis of the inhalation UR for arsenic is not included here because carcinogenicity related to inhalation exposure was eliminated from further consideration in the Tier I screening.

6.3.4 Chromium VI

6.3.4.1 Introduction

The main source of the information below is the chromium IRIS documentation [EPA, 2012a] and supporting information [EPA, 1998], plus the New Jersey Department of

Environmental Protection (NJDEP) [NJDEP, 2009] for the oral SF. Please consult those sources for further details and primary references.

Chromium is approximately the 15th most common element in the Earth's crust (http://en.wikipedia.org/wiki/Abundance_of_elements_in_Earth's_crust). The forms of chromium in the environment of most interest in terms of toxicity are compounds containing trivalent (CrIII) and hexavalent chromium (CrVI). These forms have different toxicity endpoints and mechanisms. Chromium is also an essential micronutrient for humans (see the Non-Site Related Exposures to and Nutritional Essentiality of COPCs section below). As CrIII was eliminated in the Tier I screening, the focus here is upon the more-toxic CrVI. Further, the focus is upon particulates for the RfC (as this distinction from soluble compounds is made in IRIS). Note that this HHRA assumes a standard 6:1 CrIII/VI ratio, as no information has been provided otherwise by Chino. Thus, any site or reference area chromium concentrations are reduced accordingly to evaluate risks from CrVI only.

Chromium in used in the chemical industry, in stainless-steel and other alloys, and is used for chromium plating. Aside from geologic sources, CrVI in the environment is largely man-made, and is the result of contamination by industrial sources. Examples of commonly used CrVI compounds include ammonium chromate, calcium chromate, potassium chromate, potassium dichromate, and sodium chromate.

6.3.4.2 Non-Cancer Chronic Toxicity

IRIS [EPA, 2012a] lists an oral RfD for CrVI. There is no IRIS RfC; thus the HEAST [EPA, 1997] value is used here, in accordance with the EPA RSL tables [EPA, 2012c].

Oral Exposure

The primary reference used by IRIS (MacKenzie et al., 1958) could not be located for review. The following information is based upon the IRIS summary [EPA, 2012a] and the toxicological review [EPA, 1998].

There is evidence that CrVI causes toxic effects via oral exposure in humans. However, a NOAEL could not be determined from the existing studies. A rat study was therefore used as the basis for the RfD. Groups of eight male and eight female rats were supplied with drinking water containing 0.45 to 11.2 mg/L CrVI (as K_2CrO_4) for 1 year. The control group received distilled water. A second experiment involved three groups of 12 male and 9 female rats. One group was given 25 mg/L CrVI (as K_2CrO_4), a second received 25 ppm CrIII in the form of chromic chloride, and the controls again received distilled water. No significant adverse effects were seen in appearance, weight gain, or food consumption, and there were no pathologic changes in the blood or other tissues in any treatment group. The rats receiving 25 ppm of CrVI as K_2CrO_4 showed an approximate 20% reduction in water consumption. Based upon the body weight of the rat (0.35 kg) and the average daily drinking water consumption for the rat (0.035 l/d), this dose was converted to give an adjusted NOAEL of 2.5 mg/kg-d.

For the rats treated with 0.45-11.2 ppm ppm in drinking water, blood was examined monthly, and tissues (livers, kidneys, and femurs) were examined at 6 months and 1 year.

Spleens were also examined at 1 year. The 25 ppm groups (and corresponding controls) were examined similarly, except that no animals were killed at 6 months. A rise in tissue CrVI concentrations was noted in rats treated with more than 5 ppm. The authors stated that "apparently, tissues can accumulate considerable quantities of chromium before pathological changes result." In the 25 ppm treatment groups, tissue concentrations of chromium were approximately 9 times higher for those treated with CrVI than for the CrIII group.

An UF of 300 accounts for two 10-fold decreases for both the expected interhuman and interspecies variability in the toxicity of the chemical in lieu of specific data, and an additional factor of 3 to compensate for the less-than-lifetime exposure duration of the principal study. A modifying factor (MF) of 3 accounted for "concerns raised by the study of Zhang and Li (1987)". This study was in Chinese and could not be reviewed; thus the "concerns" are unclear. Confidence in the RfD was low.

Inhalation Exposure

Occupational exposure to chromium compounds has been studied in the chromate production, chrome-plating and chrome pigment, ferrochromium production, leather tanning, chrome alloy production, and gold mining industries. The only industry that is relevant for particulate exposure (as opposed to chemical compound or fume exposure) is mining; however, this study focused upon cancer mortality. Therefore, EPA relied upon animal data for the RfC.

Glaser et al. (1985, 1990) examined respiratory effects following exposures to CrVI. Only Glaser et al. 1985 [Glaser, U., Hochrainer, D., Kloppel, H., and Kuhnen, H., 1985] could be retrieved for review. Glaser et al. exposed 5-week-old male rats to sodium dichromate at concentrations ranging from 0.025 to 0.2 mg CrVI/m^3, 22 hr/d in subacute (28 d) or subchronic (90 d) protocols. Chromium-induced effects occurred in a dose-dependent manner. Lung and spleen weights were significantly increased after both subacute and subchronic exposures at concentrations greater than 0.025 mg/m^3. Differences in the mean total serum immunoglobulin were also significant at exposures above 0.025 mg/m^3, while exposures to aerosol concentrations greater than 0.1 mg/m^3 resulted in depression of the immune system stimulation. Bronchoalveolar lavage fluid (BALF) cell counts were significantly decreased following subchronic exposure to levels above 0.025 mg/m^3. The number of lymphocytes and granulocytes showed a significant increase in the lavage fluids of the subacute and subchronically exposed groups. The spleen T-lymphocyte subpopulation was stimulated by subchronic exposure to 0.2 mg/m^3 chromium, and serum contents of triglycerides and phospholipids differed significantly from controls at this concentration.

According to EPA [EPA, 1998] Glaser et al. (1990) exposed 8-week-old male rats to sodium dichromate at 0.05 to 0.4 mg /m^3, 22 hr/d, 7 d/wk for 30 to 90 d. Chromium-induced effects occurred in a strong dose-dependent manner. The authors observed obstructive respiratory dyspnea and reduced body weight following subacute exposure at the higher dose levels. The mean white blood cell count was increased at all doses and was related to significant dose-dependent leukocytosis following subacute exposures. Mean lung weights were significantly increased at exposure levels of 0.1 mg/m^3

following both subacute and subchronic exposures. Accumulation of macrophages was seen in all of the exposure groups. Focal inflammation was observed in the upper airways following the subchronic exposure, and albumin and lactate dehydrogenase (LDH) in BALF were increased following the exposure. The authors concluded that CrVI inhalation induced pneumocyte toxicity and suggested that inflammation is essential for the induction of most chromium inhalation effects.

EPA used the benchmark concentration (BMC) approach of Malsch et al. [Malsch, P. A., Proctor, D. M., and Finley, B. L., 1994] to develop its RfC for particulates. BMCs for lung weight, LDH in BALF, protein in BALF, albumin in BALF, and spleen weight were developed based upon the Glaser et al. stuides. The BMC was defined as the 95% lower confidence limit on the dose corresponding to a 10% relative change in the endpoint compared to the control. Dose-effect data were adjusted to account for discontinuous exposure (22 hr/d) and a maximum likelihood model was used to fit continuous data to a polynomial mean response regression, yielding maximum likelihood estimates of 0.036 to 0.078 mg/m^3 and BMCs of 0.016 to 0.067 mg/m^3. Dosimetric adjustments and EPA UFs were applied to determine a RfC based upon the following equation:

$$RfC = BMC \times RDDR/UF_A \times UF_F \times UF_H$$

where: RfC is the inhalation reference concentration; BMC is the benchmark concentration; RDDR is the regional deposited dose ratio to account for pharmacokinetic differences between species; UF_A is a threefold uncertainty factor to account for pharmacodynamic differences not addressed by the RDDR; UF_F is a threefold uncertainty factor to account for extrapolating from subchronic to chronic exposures; and, UF_H is a 10-fold uncertainty factor to account for the variation in sensitivity among members of the human population.

The RDDR factor is incorporated to account for differences in the deposition pattern of inhaled CrVI dusts in the respiratory tract of humans and the rat test animals. The RDDR of 2.1576 was determined based upon the mass median aerodynamic diameter and the geometric standard deviation of the particulates. Application of the total uncertainty factor of 300 and the RDDR of 2.1576 to the BMC generated by Malsch et al. results in an RfC of 1.0E-4 mg/m^3 for inhalation of CrVI particulates. Confidence in the RfC was medium.

6.3.4.3 Carcinogenicity

There is no IRIS SF for CrVI; EPA states "the oral carcinogenicity of Cr(VI) cannot be determined. No data were located in the available literature that suggested that Cr(VI) is carcinogenic by the oral route of exposure". However, the RSL tables [EPA, 2012c] list an oral SF developed by the NJDEP [NJDEP, 2009].

There is little evidence of ingested CrVI carcinogenicity in humans. Therefore, NJDEP used data from a recent NTP study of sodium dichromate dihydrate in rats and mice [NTP, 2008] to derive a SF. Sodium dichromate dihydrate in solution yields the dichromate ion that exists in equilibrium in solution with the chromate ion. According to NJDEP, the results of the NTP study are therefore applicable to the cancer risk assessment of CrVI via ingestion. NTP concluded that the study provides "clear evidence

of carcinogenicity" in male and female mice and rats, based upon benign and malignant tumors in mouse small intestine and rat oral mucosa. The mouse was selected by NJDEP as the most sensitive species, and the human cancer slope factor was developed based upon benchmark dose (BMD) modeling and linear extrapolation below a point of departure (POD; as opposed to application of a linearized multistage model, which is the case with most EPA SFs).

The NTP study exposed male and female rats and mice to sodium dichromate in their sole source drinking water. Male mice were supplied with drinking water containing 0, 14.3, 28.6, 85.7, or 257.4 mg/L for 2 years. Female mice were supplied with 0, 14.3, 57.3, 172, or 516 mg/L for 2 years.

The SF, as applicable to intestinal tumors in male mice, was calculated by linear extrapolation through zero from a POD that was the lower confidence bound on an estimated BMD. The CrVI dose was obtained by multiplying the sodium dichromate dihydrate dose by 0.35, which is the fraction of the sodium dichromate dihydrate molecular weight contributed by chromium. The animal dose corresponding to a 1.0E-06 cancer risk to the dose corresponding to the same risk in humans was estimated using allometric scaling. For the slope derived from the male mouse data, the slope ranges from 3.0E-01 to 5.0E-01 per mg/kg-d. The higher (more conservative) value was chosen as the oral SF. NJDEP did not assign a qualitative judgement of the degree of uncertainty associated with this value. They considered that further adjustment (beyond linear extrapolation below the POD) for a mutagenic MOA was not considered appropriate. However, McCarroll et al. [McCarroll, N., Keshava, N., Chen, J., Akerman, G., Kligerman, A., and Rinde, E., 2010] of EPA considered that there is sufficient evidence of a mutagenic MOA, and thus consider application of an adjustment for children appropriate for oral CrVI. This functionally raises the oral SF for children to 1.27 per mg/kg-d. A confidence level was not provided.

A summary of the basis of the inhalation UR for CrVI is not included here because carcinogenicity related to inhalation exposure was eliminated from further consideration in the Tier I screening.

6.3.5 Cobalt

6.3.5.1 Introduction

Cobalt toxicity values are not listed in IRIS [EPA, 2012a].The main source of the information below is the cobalt PPRTV documentation [EPA, 2008]. Please consult that report for further details and primary references.

Cobalt is approximately the 30th most common element in the Earth's crust (http://en.wikipedia.org/wiki/Abundance_of_elements_in_Earth's_crust). Before the 19th century, the predominant use of cobalt was as a blue pigment in ceramics, glass, and other uses. The main current application of cobalt is as a component of metal alloys. Cobalt oxide is used in some types of batteries. Several cobalt compounds are used in chemical reactions as oxidation catalysts. Radioactive cobalt-60 (not a concern at the LIU) is useful as a gamma ray source in medicine (e.g., for radiation treatment of cancer),

and is used in some nuclear weapons. Cobalt is a constituent of the essential vitamin B12, but is not considered an essential element *per se*.

6.3.5.2 Non-Cancer Chronic Toxicity

Oral Exposure

Indicators of human health effects following oral exposure to cobalt include increased erythrocyte production and hemoglobin levels, decreased iodine uptake by the thyroid gland, elicitation of dermatitis in sensitized individuals and cardiomyopathy. Observations in humans for effects upon the heart, blood and the thyroid gland are supported by results of studies in animals. Other effects, including neurobehavioral, developmental and testicular toxicity have been observed in animals at relatively high doses [EPA, 2008].

Due to numerous issues associated with studies of the effects above, EPA chose thyroid toxicity as the critical effect for derivation of the provisional RfDs. Cobalt-induced polycythemia and decreased iodine uptake by the thyroid were reversible following relatively short-term exposure in humans, however supporting studies indicate the potential for more severe thyroid effects. An exposure of 1.0 mg/kg-d, which resulted in decreased iodine uptake, was determined to be a LOAEL based upon the study of Roche and Layrisse (1956). This study could not be retrieved for review. According to EPA [EPA, 2008], treatment of 12 patients (with normal thyroids) with 150 mg cobalt chloride/d (equivalent to 1.0 mg cobalt/kg-d, assuming a body weight of 70 kg) for 2 weeks resulted in a greatly reduced uptake of 48-hour radioactive iodine by the thyroid when measured after 1 week of exposure, with uptake nearly abolished completely by the second week of exposure to cobalt. When cobalt treatment was discontinued, iodine uptake returned to pre-treatment reported values. No other clinical details were provided for the human subjects.

A composite UF of 3000 was applied to this LOAEL to result in a RfD of 3E-04 mg/kg-d. An UF of 10 for extrapolation from subchronic to chronic duration was applied because the critical effect was chosen from a principal study of a relatively short duration (2 weeks) of oral exposure in humans. An UF of 10 for LOAEL to NOAEL extrapolation was applied because the POD is based upon a LOAEL. An UF of 10 was applied due to the lack of data regarding inter-individual human variability or information on sensitive subpopulations. Specifically, because the critical study (Roche and Layrisse, 1956) for oral cobalt was based upon healthy adults, an UF of 10 was applied to protect sensitive human populations. Confidence in the RfD was low-to-medium.

Inhalation Exposure

Respiratory effects are sensitive endpoints of inhaled cobalt. Symptoms of respiratory tract irritation and altered pulmonary function have been widely reported in workers exposed to cobalt-containing compounds. The study by Nemery et al. [Nemery, B., Casier, P., Roosels, D., Lahaye, D., and Demedts, M., 1992] provided the strongest basis for derivation of a RfC. This was a cross-sectional study of cobalt exposure and respiratory effects in diamond polishers who were primarily exposed to metallic cobalt-containing dust. The values obtained from personal air samples from the Nemery et al. study indicated a NOAEL of 5.3 $\mu g/m^3$ and a LOAEL of 15.1 $\mu g/m^3$ for effects upon pulmonary function (e.g.; forced expiratory volume, forced vital capacity, and forced expiratory flow) and an increased prevalence of symptoms of respiratory tract irritation (e.g.; nose/throat irritation, cough, phlegm, dyspnea). This study demonstrated a dose-effect relationship with regard to lung function that correlated with urinary cobalt levels, after adjusting for effects of smoking and gender.

The NOAEL for occupational exposure was adjusted for continuous exposure as follows:

5.3 $\mu g/m^3$ x (10 m^3/d / 20 m^3/d) (5d / 7d) = 1.9 $\mu g/m^3$.

Dividing this value by a composite UF of 300 yields a RfC of 6.0E-06 mg/m^3 for metallic cobalt. The composite UF of 300 is composed of three uncertainty factors: 3 to account for extrapolating from an assumed subchronic exposure duration to a chronic exposure duration; 10 for database insufficiencies; and, 10 for human inter-individual variability. Confidence in the RfC was medium to low.

6.3.5.3 Carcinogenicity

According to EPA [EPA, 2008], there is inadequate information to assess the carcinogenicity of cobalt via oral exposure. A summary of the basis of the inhalation UR for cobalt is not included here because carcinogenicity related to inhalation exposure was eliminated from further consideration in the Tier I screening.

6.3.6 Manganese

6.3.6.1 Introduction

The main source of the information below is the manganese IRIS documentation [EPA, 2012a] and supporting information. Please consult that source for further details and primary references.

Manganese is approximately the 12th most common element in the Earth's crust (http://en.wikipedia.org/wiki/Abundance_of_elements_in_Earth's_crust). Manganese is used in steel production and aluminum alloys. Methylcyclopentadienyl manganese tricarbonyl is used as an additive in unleaded gasoline to boost octane rating and reduce engine knocking. Manganese oxides are used as a reagent in organic chemistry, in glassmaking, as pigments, and in batteries. Manganese is an essential element for humans (see the Non-Site Related Exposures to and Nutritional Essentiality of COPCs section below); this is considered in the development of the oral RfD.

6.3.6.2 Non-Cancer Chronic Toxicity

Oral Exposure

As opposed to the other COPCs, the RfD for manganese was apparently not explicitly derived based upon toxic effects, although there may be evidence of neurological effects at high doses (and such effects occur with inhalation exposure; see below). IRIS [EPA, 2012a] states "the reference dose is estimated to be an intake for the general population that is not associated with adverse health effects; this is not meant to imply that intakes above the reference dose are necessarily associated with toxicity. Some individuals may, in fact, consume a diet that contributes more than 10 mg Mn/day [0.14 mg/kg-d) without any cause for concern". The exact basis for the RfD is unclear from IRIS, as three sources (Freeland-Graves et al. 1987, National Research Council [NRC] 1989, and World Health organization 1973) are provided. From the IRIS text, it can be inferred that the NRC report on recommended daily allowances [NRC, 1989] was the source of the 10 mg/d value; i.e., "in view of the remarkably steady tissue concentrations of manganese in the U.S. population. . .and the low toxicity of dietary manganese, an occasional intake of 10 mg/day by adults can be considered safe" (p. 233). The NRC then states "to include an extra margin of safety, however, the subcommittee recommends a range of manganese intake from 2 to 5 mg/ day for adults" (p.233). IRIS implies that the NRC recommended values may be too low, and states "depending on individual diets, however, a normal intake may be well over 10 mg Mn/day, especially from a vegetarian diet".

Based upon these sources, EPA concluded that an appropriate RfD for manganese is 10 mg/d, or 0.14 mg/kg-d. This value applies to manganese in food. For manganese in water or soil, EPA applies a modifying factor of 3, which results in a RfD of 0.024 mg/kg-d. EPA's [EPA, 2012a] justifications for this factor include: There is increased uptake of manganese from water in fasted individuals; there are possible adverse health effects associated with a lifetime consumption of drinking water containing about 2 mg/L of manganese; "although toxicity has not been demonstrated, there is concern for infants fed formula that typically has a much higher concentration of manganese than does human milk. . .if powdered formula is made with drinking water, the manganese in the water would represent an additional source of intake"; and, there is evidence that compared to adults neonates absorb more manganese from the gastrointestinal tract, they are less able to excrete absorbed manganese, and the absorbed manganese more easily passes the blood-brain barrier. However, note that current US Department of Agriculture Dietary Reference Intakes (DRIs) for manganese [USDA, 2012] range from 0.003 mg/d for infants to 2.6 mg/d for lactating females, with an average across adult males and females of 2.0 mg/d or 0.06 mg/kg-d. It is difficult to determine from these disparate values whether the manganese water/soil RfD is appropriate in terms of striking a balance with nutritional requirements of adults, but it is likely protective against toxic effects.

Although EPA publishes a 'food' RfD in IRIS, the appropriate RfD applied here is 0.024 mg/kg-d, as food exposures are not of interest. Confidence in the oral RfD was medium.

Inhalation Exposure

The manganese RfC in IRIS [EPA, 2012a] is based upon the study of Roels et al. [Roels, H. A., Ghyselen, P., Buchet, J. P., Ceulemans, E., and Lauwerys, R. R., 1992] who

conducted a cross-sectional study of 92 male workers exposed to manganese dioxide (MnO^2) dust in a Belgian alkaline battery plant. The exposed group had been exposed to MnO^2 for an average of 5.3 years. Occupational-lifetime integrated exposure to manganese was estimated for each worker by multiplying the current airborne manganese concentration for the worker's job classification by the number of years for which that classification was held and adding the resulting (arithmetic) products for each job position a worker had held. The geometric mean occupational-lifetime integrated respirable dust (IRD) concentration was 0.793 mg/m^3 x years (range: 0.040 to 4.433 mg mg/m^3 x years), with a geometric standard deviation of 2.907 mg/m^3 x years. A self-administered questionnaire focused upon occupational and medical history, neurological complaints, and respiratory symptoms. Workers performed worse than controls on several measures of neurobehavioral function. Visual reaction time was consistently and significantly slower. Five measures of eye-hand coordination (precision, percent precision, imprecision, percent imprecision, and uncertainty) reflected more erratic control of fine hand-forearm movement in the exposed group than in the controls, with mean scores on all five measures being highly significantly different for the two groups. EPA [EPA, 2012a] derived a LOAEL from the Roels et al. study by using the IRD concentration of MnO^2, expressed as mg manganese/m^3 x yr (based upon 8-hour TWA occupational exposures for various job classifications, multiplied by individual work histories in years). Dividing the geometric mean IRD concentration (0.793 mg/ m^3 x yr) by the average duration of the workers' exposure to MnO^2 (5.3 yr) yields a LOAEL of 0.15 mg/ m^3. A LOAEL(HEC) (human equivalent concentration) was derived by EPA by multiplying this value by 5 / 7 d and $10m^3/d$ / $20m^3/d$ to adjust from occupational to public exposure. The result was 0.05 mg/m^3.

An uncertainty factor of 1000 was applied to this LOAEL(HEC), and includes a factor of 10 to protect sensitive individuals, 10 for use of a LOAEL, and 10 for database limitations reflecting both the less-than-chronic periods of exposure and the lack of developmental data, as well as potential but unquantified differences in the toxicity of different forms of manganese. Confidence in the RfC was medium.

6.3.6.3 Carcinogenicity

According to EPA [EPA, 2012a], there is inadequate information to assess the carcinogenicity of manganese via oral exposure.

6.4 Non-Site Related Exposures to and Nutritional Essentiality of COPCs

All of the LIU COPCS are naturally-occurring, and thus can be present in soil, water, food, and air. Addtionally, chromium and manganese (plus cobalt as a constituent of vitamin B12) are considered essential nutrients; and complete absence of these nutrients can result in illness. Thus, it can be useful to qualitatively compare typical levels present in the environment, as well as levels essential for good health, to predicted exposures associated with a contaminated site. Quantitative comparisons would be complex and are not useful for this HHRA, as issues such as geographic variability, differences in diet, etc. would need to be addressed. Rather, information on non-site related exposures and

nutritional essentiality can simply provide a 'rough' context for any estimated exposures and RME risks assocated with COPCs at the LIU.

Table 26: Approximate Non-Site Related Environmental Concentrations and Dietary Intakes of COPCs

COPC	Soil (mg/kg)[1]	Reference	Water (mg/L)[2]	Reference	Diet (including supplements) (mg/d)[3]	Reference	Dietary Reference Intakes (mg/d)[3]	Reference
Aluminum	4300 to 100000	[Kabata-Pendias, A. and Pendias, H., 1984]	0.07	[Langmuir, D., Chrostowski, P., Vigneault, B., and Cheney, R., 2005]	3.4 to 9.0 (5000)[4]	[ATSDR, 2008a]	NA	NA
Arsenic	0.1 to 93		3.0E-05 to 43		2.3E-02 to 7.2E-02	[IOM, 2001]	NA	NA
Chromium	1.0 to 1500		5.0E-04 to 3.8		1.3E-02 to 5.4E-02	[IOM, 2001]	2.0E-04 to 4.5E-02	[USDA, 2012]
Cobalt	0.3 to 50		0 to 9.0E-03	[ATSDR, 2004]	0.01[5]	[ATSDR, 2004]	NA	NA
Manganese	7.0 to 3000		4.0E-03 to 0.7	[ATSDR, 2008b]	0.52 to 11	[IOM, 2001]	3.0E-03 to 2.6	[USDA, 2012]

NA: not applicable or available
1: Ranges include all types of soils nationwide
2: All groundwaters except for aluminum (surface waters)
3: The ranges of dietary intakes and recommended dietary reference intakevalues for essential nutrients (if given) are reflective of age, gender, and other factors. Dietary reference intakes include recommended daily allowances (RDAs). These are intake doses, not absorbed doses.
4: Value in parentheses represents upper amount ingested via antacid products.
5: Cobalt is a component of vitamin B12, but is not listed as "essential" *per se* here as it does not have a dietary reference intake.

6.5 Risk Characterization

6.5.1 Overview

In the Tier II risk characterization, site-related COPC exposures (using 95% UCLs as EPCs) and toxicity values are combined to produce estimates of non-carcinogenic hazard and ILCR. These estimates are then compared with 'acceptable' levels, as determined by regulatory guidance, precedent, and discussion among involved parties.

Similar considerations applied in Tier I with regard to estimation of HQs/HIs and ILCRs apply here. HQs above 1.0 (i.e., the estimate intake level exceeds the RfD) are of potential concern. The potential for additive non-carcinogenic effects across two or more COIs is evaluated in the HHRA only in cases where the toxic effects of the COIs are similar. The sum of two or more HQ values is referred to as a Hazard Index (HI). A HI value exceeding 1.0 may be of concern even if the HQs for all individual COIs are below 1.0, but only if the individual COIs have similar toxicological endpoints (see Table 8).

NMED has defined 1E-05 (0.00001) as a target for development of its Soil Screening Levels (SSLs) [NMED, 2012]. A 1E-05 ILCR is used as a target level for Tier II. The two potential carcinogenic COPCs are arsenic and CrVI. ILCRs from oral exposure for these COPCs are not added here because the MOAs and the bases of their SFs are very different [EPA, 1989;EPA, 2005a]. See the Toxicity Assessment section for further details.

6.5.2 Results

6.5.2.1 Hazard Quotient Results

Results below are presented in Table 27, Table 28, and Table 29 as individual COPC HQs, plus COPCs are combined as appropriate in terms of toxic effects (see Table 8). Note that cardiovascular effects are only attributable to arsenic as a COPC, so combined effects (as HIs) are not represented. Scenarios A, B, and D were screened out in Tier I, as was surface water; and thus are not represented. In all cases, "NA" in tables refers to a lack of relevant COPC data (either not detected or not analyzed) or missing toxicity values; thus no HQ is estimated. "NR" means "not relevant".

Bold values exceed a HQ of 0.1, and ***bold/italic*** values exceed a HQ of 1.0. Only COPCs with HQs exceeding 0.1 are summed, and then only if toxicologically relevant (per Table 8). Only individual COPCs with HQs greater than 1.0 or toxicologically relevant combinations of COPCs (HIs) greater than 1.0 are of interest in terms of noncarcinogenic hazard.

Scenario C (residence) is the only scenario evaluated that included children. Children tend to exceed chronic toxicity criteria (e.g., greater than a HQ of 1.0) to a greater degree than adults, due to lower body weight, higher soil contact, and other considerations. In all cases, the estimated HQs for children exceeded those of adults. However, both children and adult HQs are presented for context. Interpretation of results follows the tables.

Table 27: Tier II Scenario C (Residence) Hazard Quotients (adult)

COI	Soil Ingestion	Dermal Absorption	Soil Ingestion + Dermal Absorption	Dust Inhalation	*Total*	Exceeds Target HQ/HI?
Aluminum	8.5E-05	1.9E-03	1.9E-03	1.7E-02	1.9E-02	NO
Arsenic	9.4E-03	1.3E-03	1.1E-02	2.4E-03	1.3E-02	NO
Chromium VI	1.4E-04	1.6E-05	1.6E-04	2.1E-04	3.7E-04	NO
Cobalt	1.3E-02	4.2E-03	1.8E-02	9.5E-03	2.7E-02	NO
Manganese	1.4E-02	7.5E-02	8.9E-02	6.5E-02	**1.5E-01**	NO
TOTAL neurotoxicity (aluminum, arsenic, cobalt, manganese)	3.7E-02	8.3E-02	**1.2E-01**	9.4E-02	**2.1E-01**	NO

Bold values exceed HQ of 0.1, and ***bold/italic*** values exceed HQ of 1.0. Only COIs with HQs exceeding 0.1 are summed for screening purposes, and then only if toxicologically relevant.

Table 28: Tier II Scenario C (Residence) Hazard Quotients (child)

COI	Soil Ingestion	Dermal Absorption	Soil Ingestion + Dermal Absorption	Dust Inhalation	*Total*	Exceeds Target HQ/HI?
Aluminum	9.0E-04	3.5E-03	4.4E-03	2.5E-02	3.0E-02	NO
Arsenic	1.0E-01	2.5E-03	1.0E-01	3.6E-03	**1.1E-01**	NO
Chromium VI	1.5E-03	2.9E-05	1.5E-03	3.2E-04	1.9E-03	NO
Cobalt	**1.4E-01**	7.9E-03	**1.5E-01**	1.4E-02	**1.7E-01**	NO
Manganese	**1.5E-01**	**1.4E-01**	**2.9E-01**	9.8E-02	**3.9E-01**	NO
TOTAL neurotoxicity (aluminum, arsenic, cobalt, manganese)	**3.9E-01**	**1.6E-01**	**5.5E-01**	**1.4E-01**	**6.9E-01**	NO

Bold values exceed HQ of 0.1, and ***bold/italic*** values exceed HQ of 1.0. Only COIs with HQs exceeding 0.1 are summed for screening purposes, and then only if toxicologically relevant.

Table 29: Tier II Scenario E (Construction) Hazard Quotients

COI	Soil Ingestion	Dermal Absorption	Soil Ingestion + Dermal Absorption	Dust Inhalation	*Total*	Exceeds Target HQ/HI?
Aluminum	6.1E-05	1.3E-03	1.4E-03	**3.2E-01**	**3.2E-01**	NO
Arsenic	6.7E-03	9.5E-04	7.7E-03	4.5E-02	5.3E-02	NO
Chromium VI	1.0E-04	1.1E-05	1.1E-04	4.0E-03	4.1E-03	NO
Cobalt	9.6E-03	3.0E-03	1.3E-02	**1.8E-01**	**1.9E-01**	NO
Manganese	1.0E-02	5.4E-02	6.4E-02	***1.2E+00***	***1.3E+00***	***YES***
TOTAL neurotoxicity (aluminum, arsenic, cobalt, manganese)	2.6E-02	5.9E-02	8.6E-02	***1.8E+00***	***1.9E+00***	***YES***

Bold values exceed HQ of 0.1, and ***bold/italic*** values exceed HQ of 1.0. Only COIs with HQs exceeding 0.1 are summed for screening purposes, and then only if toxicologically relevant. Manganese dominates total neurotoxicity, thus the other neurotoxic COPCs are not represented as "exceeds target" (see text).

6.5.2.2 *Interpretation of Hazard Quotient Results*

The defined regulatory criteria for COPCs, as previously stated, are exceeding a HQ of 1.0, or a particular COI with a HQ of between 0.1 and 1.0 in combination with other COIs with similar toxic effects exceeding a total HI of 1.0. These HQs and HIs are based upon 95% UCLs.

The results indicate that manganese is the only COPC that exceeds a HQ of 1.0, and only in Scenario E. The total manganese HQ is 1.3. The most important pathway is inhalation (about 95% of total hazard).

The manganese RfD and RfC are based upon neurotoxicity. Aluminum, arsenic, and cobalt also have potential neurotoxic effects. However, manganese contributes 68% of hazard totaled across these COPCs in Scenario E. The next highest contributor is aluminum (HQ of 0.32), largely due to inhalation.

The most important exposure factor that contributes to the elevated manganese HQ in Scenario E is the PEF. This PEF in this scenario (2.55E+06 m^3/kg) is reflective of dusty conditions as a result of construction traffic. If the PEF that is used for the other scenarios (1.34E+08 m^3/kg) is used instead, the manganese HQ drops to 0.087. Thus, the results are highly sensitive to the construction PEF and its inherent assumptions (see Appendix I: Exposure Equations).

6.5.2.3 *Incremental Lifetime Cancer Risk Results*

Results are presented in Table 30 and Table 31 as individual COPC ILCRs. As stated previously, the only COPCs that have similar effects (i.e., lung cancer) are the carcinogenic COPCs that have inhalation URs. However, inhalation routes were screened out in Tier I.

In all cases, "NA" in tables refers to a lack of relevant COI data (either not detected or not analyzed) or missing toxicity values; thus no ILCR is estimated. "NR" means "not relevant".

Bold values exceed an ILCR of 1E-06, and ***bold/italic*** values exceed an ILCR of 1E-05. Only individual COPCs with ILCRs greater than 1E-05 are of interest in terms of risk management decisions.

Interpretation of results follows the tables.

Table 30: Tier II Scenario A (Commercial Ranching) Incremental Lifetime Cancer Risks

COI	Soil Ingestion	Dermal Absorption	*Total*	Exceeds Target ILCR?
Arsenic	7E-07	1E-07	8E-07	NO
Chromium VI	3E-08	4E-09	4E-08	NO
Bold values exceed ILCR of 1E-06, and ***bold/italic*** values exceed ILCR of 1E-05.				

Table 31: Tier II Scenario C (Residence) Incremental Lifetime Cancer Risks

COI	Soil Ingestion	Dermal Absorption	Total	Exceeds Target ILCR?
Arsenic	**5E-06**	3E-07	**5E-06**	NO
Chromium VI	6E-07	3E-08	6E-07	NO
Bold values exceed ILCR of 1E-06, and ***bold/italic*** values exceed ILCR of 1E-05.				

6.5.2.4 *Interpretation of Incremental Lifetime Cancer Risk Results*

The defined ILCR decision criterion for COPCs in Tier II, as previously stated, is an ILCR greater than 1E-05. In no cases did ILCRs exceed this level. Although arsenic and chromium have different cancer endpoints via oral exposure and likely have different MOAs, and thus adding ILCRs is not recommended [EPA, 2005a;EPA, 1989]; summing these ILCRs regardless does not result in a combined ILCR exceeding 1E-05.

6.6 *Tier II Human Health Risk Assessment Summary*

A Tier II HHRA was conducted for the LIU using 95% UCLs for COPC EPCs and conservative scenarios and exposure assumptions. The intent of this was to identify the most risk-relevant scenarios and exposure pathways, and COPCs potentially important in terms of risk management decisions.

The only situation in which target hazard/risk levels was marginally exceeded was the case of manganese exposure in the future construction Scenario E (HQ=1.3). This was largely attributable to dust inhalation, and largely in turn, the amount of dust generated during construction activities (as determined by the PEF). It is possible that the other COPCs that have similar toxic effects (i.e., neurotoxicity) could contribute approximately 30% more hazard.

Comparisons with common exposure levels are useful. The air concentration of manganese that results in the inhalation HQ of 1.2 is 6.0E-05 mg/m^3. To convert this to a daily exposure level, this value can be multiplied by a standard adult inhalation rate of 20 m^3/d. This results in 1.2E-03 mg/d. This is 2 to 4 orders of magnitude lower than typical daily dietary levels (Table 26). The US Department of Agriculture Dietary Reference Intake for adult males is 2.3 mg/d [USDA, 2012]. The estimated manganese exposure level that results in the HQ of 1.2 is thus 0.05% of the recommended intake level. The LOAEL(HEC) that EPA used to develop the manganese RfC was 0.05 mg/m^3 (or 1.0 mg/d), and a total UF of 1000 was applied. It would be difficult to determine whether the estimated incremental level of manganese exposure via inhalation in Scenario E would actually result in toxic effects. Given the level of conservatism in the RfC, this seems unlikely.

Compared to reference site concentrations, manganese was only statistically elevated in surface soil compared to the STSIU/ERA data, and then only marginally according to one test (the slippage test). This test indicates a possible shift in the upper tail of the COPC distribution.

Arsenic did not exceed the ILCR threshold of 1E-05, but the ILCR was approximately 50% of the threshold (5E-06). This was largely driven by soil ingestion. It is possible, for example, given BFs that were twice those used here or other similar modifications in exposure variables that the arsenic ILCR would exceed the threshold. However, there are no obvious variable values that would warrant modification and thus cause such an increase. Arsenic has been subject to a relatively large amount of scrutiny with regard to bioavailability, as it is a common COPC at many sites, and thus a relatively high degree of confidence can be placed in the values employed here. Addtionally, as discussed in the Toxicity Assessment section, the arsenic SF is likely to be highly conservative.

LIU site surface soil arsenic concentrations were elevated compared to the STSIU/ERA reference data (using all three statistical tests), but not to the LIU reference data. It is possible that arsenic is 'naturally' elevated in the LIU compared to other Chino areas. The RI [Arcadis, 2012] presents extensive discussion of the local geology. Regardless, it does not appear that arsenic warrants concern at the LIU.

Previous HHRAs at the Chino site have compared 95% UCL-driven hazards and risks with "central tendency" (i.e., mean)-driven hazards and risks. As there was only one situation where hazard was elevated, and that hazard is largely attributable to the PEF, it is not informative to present central tendency hazard/ILCR estimates here. It is also not informative to present risks associated with reference areas, as in most cases there was no statistical difference between site and reference area concentrations. In general, however, estimated reference area risks are likely to be similar to or perhaps in some cases higher than site-related risks.

7 Uncertainty Assessment

There are numerous sources of uncertainty associated with the results of the HHRA. In general, the RME approach ensures that the estimates are biased toward conservatism (i.e., being protective), and thus estimates of hazard and risk are protective. For example, use of maximum detected values and 95% UCLs for EPCs is conservative, as are many of the exposure assumptions and all of the toxicity values. However, potential exceptions are noted below.

Screening Approach

- The HHRA used a site-specific screening approach (Tier I), as described previously. It is possible that a different set of COPCs and scenarios would pass another type of screen (e.g., RSLs). The assumptions in the Tier I screen could also be different; e.g., use of 1E-06 as a target ILCR rather than 1E-05. Regardless, use of a slightly different screening method would not substantially affect the COPCs and scenarios evaluated in Tier II, nor would it affect the final conclusions.

CSM

- The defined scenarios could differ. The scenarios were based upon current and expected future land uses, but other scenarios could exist. For example, it may be possible that industrial development other-than mining could occur in the future. However, it is expected that the defined scenarios are conservative and thus protective compared to other potential scenarios. Conversely, it may be possible that receptors that are exposed at the LIU may be exposed at other Chino IUs or other sources of COPCs. It would be difficult to determine the relative impact of such a situation without a site-wide HHRA.
- Food pathways were not included in the HHRA. In the HWCIU and STSIU HHRAs [Neptune, 2008;Gradient, 2008], food pathways (e.g., home-grown produce, chickens, beef, etc.) were evaluated. In the case of the LIU, NMED has determined that modeling potential exposures related to home-raised foods and

game would provide limited information for risk management in the LIU. NMED has decided not to pursue the foodstuff pathways in the LIU HHRA because of the low likelihood that future residents would engage in extensive agricultural activities or gather extensive site-related game, and because cultivation of produce will likely require appreciable amendments for productive garden soil, due to the poor quality of existing soil. If such activities exist in the future, then exposures may be underestimated.

- Groundwater was not evaluated here, as it is addressed under a different regulatory structure. If future residential development occurs at the LIU, then groundwater should be evaluated to ensure that residents are protected.

Site Data

- It is possible that sampling did not capture site COPC concentrations in an accurate and precise fashion. This could result in EPCs being over- or underestimated. The sources of data employed were at varying points in time, and the quantity of data varied. This reflects the reality of data collection at a complex site. However, the design of sampling and the statistics applied for contaminated sites was standard for RIs, and quality control samples were used to assure that analytical results are within acceptable levels of precision and accuracy. Given the multiple levels of inherent conservatism in the RME approach, it is unlikely that additional or different sampling would be productive. However, if concerns remain regarding the impact of Santa Rita pit operations, or with regard to site concentrations versus reference areas; then additional sampling may be warranted.
- No information was available on the site-related concentration of CrVI versus CrIII. As previously stated, EPA [EPA, 2012a] assumes that the inhalation UR value has a ratio of CrIII to CrVI of 6:1 in air. This ratio has been used in previous HHRAs at the site (as well as numerous other HHRAs in the US), and was assumed here in lieu of site-specific information. If the actual relative concentration of CrVI is higher, then this will proportionally affect risk values. However, the concentration of CrVI would need to be increased by a factor of approximately 1.5 orders-of-magnitude in order to exceed the 1E-05 risk level in the residence (C) scenario. This is unlikely.

Modeling

- Some assumptions regarding fate-and-transport and exposure manifested in the simple models here may not be applicable. For example, the calculations that result in the PEFs and subsequent estimate of dust exposure could under- or overestimate actual exposures. The best way to confirm the results of these models would be to conduct, for example, field measurements of dust concentrations, and biomonitoring of COPCs in receptors. Again, given the multiple levels of inherent conservatism in the RME approach, it is unlikely that this would be productive.

- The construction Scenario E was the only scenario where target hazards/risks were exceeded in Tier II. In this scenario, manganese hazards are driven largely by exposure via dust inhalation. The major uncertainty in the exposure assessment for this scenario relates to the model used to estimate ambient levels of atmospheric dust. Dust levels for the construction scenario were modeled based on dust generated by vehicular traffic on unpaved roadways. While this is a credible mechanism, the 1-hr modeled value is more than twice the Federal 24-hour PM_{10} standard of 0.15 mg/m^3, and may therefore represent extreme conditions that are unlikely to reflect actual chronic dust levels over the 1-year exposure period. Because the model reflects hypothetical construction activities, refinement of the model with site-specific inputs or benchmarking with field data is not presently feasible. Collection of ambient air respirable particulate data during any future construction would confirm actual dust loading values.
- Multiple interacting toxic effects (that could be additive, synergistic, or antagonistic) from multiple COPCs were not evaluated. As described in the Toxicity Assessment section, EPA typically assumes one critical effect per toxicant; an assumption that was not challenged here. It is possible, however, that effects other-than the critical effect could be important for any particular COPC, or in combinations of COPCs. Such interactions could occur in absorption, distribution, or other phases in the human body. This would be highly complex to address, and is beyond the scope of this HHRA.
- ILCRs were only summed for the inhalation route carcinogens, for which lung cancer is the common tumor endpoint. Arsenic and CrVI oral exposure cancer risks were not summed *a priori* because, as described in the Toxicity Assessment section, the MOAs and the bases of the SFs are very different. The arsenic oral SF is based upon studies of a specific type of skin cancer in humans, with appreciable uncertainty regarding the MOA and potential existence of a threshold. The CrVI oral SF is based upon a BMD analysis of intestinal tumors in rodents, and there appears to be disagreement as to the MOA. The SFs were derived in such different ways that addition would not be mathematically meaningful. However, the NMED target of 1E-05 is not exceeded even if arsenic and CrVI oral cancer risks are summed.

<u>Exposure and Toxicity Variables</u>

- The relative bioavailability factors (oral and dermal) employed in Tier II are subject to uncertainty. Regardless, nearly all metals bound to soil are marginally bioavailable. Addtionally, interactions between COPCs (e.g., arsenic and aluminum) in a soil substrate may limit bioavailability further. Thus, the estimates employed are not likely to underestimate bioavailability.
- Professional judgment was employed regarding proportion of time spent in upland (soil) areas versus tributaries (sediment). Based upon calculation checks, changing these proportions did not change the results appreciably.

- Toxicity values were missing for some COPCs, either because EPA has made a judgement that particular effects (especially carcinogenicity) are not likely to be an issue, or simply because insufficient information exists to derive toxicity values. It is difficult to judge the impact that this source of uncertainty has upon the HHRA, especially given the high degree of conservatism that exists in the published values.
- As noted in the Toxicity Assessment section, there is a large degree of uncertainty associated with many toxicity values (RfDs, RfCs, SFs, URs). Due to multiple UFs and the assumption that the carcinogens have no threshold; it is unlikely that any of the values for the COPCs underestimates toxicity. It is likely, however, that some of these values include an excessive degree of conservatism.

8 Preliminary Remediation Goals

The only situation where hazard/risk was elevated was manganese via dust inhalation in the future construction Scenario E, and this was largely attributable to a high PEF. Manganese site concentrations do not appear to be elevated above the LIU reference data, and marginally elevated above the STSIU/ERA reference data. Therefore, it is not informative to estimate preliminary remediation goals at this time.

9 Conclusions

A baseline HHRA has been conducted for the LIU to evaluate the potential for adverse human health effects associated with historical mining operations. The HHRA provides the best information possible to make informed and expedient risk-based decisions regarding the LIU.

The HHRA followed a two-tiered approach. The screening-level Tier I assessment assessed maximum detected concentrations of COIs in exposure equations that included conservative exposure and chemical toxicity assumptions. This Tier I assessment identified COPCs carried forward to the Tier II HHRA, which included refined assumptions.

Based upon the Tier I screening; the COPCs included aluminum, arsenic, CrVI, cobalt, and manganese. Comparisons of LIU site concentrations of these COPCs with concentrations at the LIU reference area plus the STSIU/ERA reference area revealed little statistical differences between historically impacted areas and relatively non-impacted areas. The only potential issue from a human health perspective may be nervous system effects related to manganese concentrations in soils in a future construction scenario. However, the estimated elevated HQ is likely due to highly conservative assumptions regarding the quantity of dust generated by vehicle traffic on unpaved roads used in the exposure assessment. Manganese site concentrations do not appear to be elevated above the LIU reference data, and marginally elevated above the STSIU/ERA reference data. Therefore, it is not informative to estimate preliminary remediation goals at this time.

If the estimated risks or levels of uncertainty are unacceptable to the involved parties, then it may be informative to conduct a more detailed probabilistic (i.e., Monte Carlo simulation) assessment to identify the degree of conservatism associated with the Tier II assessment and to identify important sources of uncertainty. This may also include further collection or analysis of site data.

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Appendix I: Exposure Equations

These equations are based upon Section 6 of EPA [EPA, 1989].

A distinction is made here between 'intake', which is defined as the rate at which a COI is taken into the body, and absorbed dose, which is the amount of the COI that is absorbed into the bloodstream [EPA, 1989].

The general equation for intake [EPA, 1989] is:

$$\text{Intake} = \frac{C \times CR \times EF \times ED}{BW \times AT}$$

where,

Intake = chronic daily COI intake (mg/kg body weight/d)

C = COI concentration in exposure medium (e.g., mg/kg soil, mg/L water)

CR = contact rate (e.g., mg soil/d, L water/d)

EF = exposure frequency (d/yr)

ED = exposure duration (yr)

BW = body weight (kg)

AT = time over which exposure is averaged for experiencing adverse effect (d)

Separate intake calculations are performed for adults and children when evaluating noncarcinogenic effects because the averaging time over which effects are assessed is equal to the exposure duration [EPA, 1989]. However, because cancer risk is expressed as a probability averaged over a lifetime and intake is commonly higher for young children than adults, exposure as a child and adult is integrated in intake calculations for carcinogenic effects. The general intake equation is modified for evaluating carcinogenic effects according to:

$$\text{Intake}_{carc} = \frac{C \times CR \times \{(EF\_c \times ED\_c / BW\_c) + (EF\_a \times ED\_a / BW\_a)\}}{AT}$$

where, the designations "c" and "a" refer to child and adult values, respectively.

The addition of a term for the efficiency of absorption across an exchange boundary to either intake equation results in an equation for absorbed dose. The following media-specific and route-specific equations for absorbed dose are used in the risk assessment. The equations are shown for a single receptor. Modification for application to carcinogenic effects for an exposure period beginning at birth is done in the same manner as that shown for the general intake equation above.

Ingestion of Soil/Sediment

$$\text{Dose} = \frac{C\_s \times IR\_s \times BF\_ing \times FS \times EF \times ED \times CF}{BW \times AT}$$

where,

Dose = chronic daily absorbed dose, adjusted for body weight (mg/kg/d)

C_s = COI concentration in soil (mg/kg)

IR_s = ingestion rate of soil (mg soil/d)

BF_ing = relative bioavailability fraction for soil ingestion (dimensionless)

FS = fraction of ingestion associated with site (dimensionless)

EF = exposure frequency (d/yr)

ED = exposure duration (yr)

CF = units conversion factor (1×10^{-6} kg/mg)

BW = body weight (kg)

AT = averaging time (d)

Surface Water Ingestion

$$\text{Dose} = \frac{C\_w \times IR\_w \times EF \times ED}{BW \times AT}$$

where,

Dose = chronic daily absorbed dose, adjusted for body weight (mg/kg/d)

C_w = COI concentration in surface water (mg/L)

IR_w = ingestion rate of water (L/d)

EF = exposure frequency (d/yr)

ED = exposure duration (yr)

BW = body weight (kg)

AT = averaging time (d)

Dermal Contact with Soil/Sediment

$$\text{Dose} = \frac{C\_s \times BF\_derm \times FS \times \sum_i (DSA_i \times DSAF_i) \times EF \times ED \times CF}{BW \times AT}$$

where,

Dose = chronic daily absorbed dose, adjusted for body weight (mg/kg/d)

C_s = COI concentration in soil (mg/kg)

BF_derm = relative bioavailability fraction for soil dermal contact (dimensionless)

FS = fraction of soil dermal contact associated with site (dimensionless)

DSA_i = dermal surface area of body part *i* (cm^2)

$DSAF_i$ = dermal soil adherence factor of body part *i* (mg/cm^2- event)

EF = exposure frequency (d/yr)

ED = exposure duration (yr)

CF = units conversion factor (1×10^{-6} kg/mg)

BW = body weight (kg)

AT = averaging time (d)

Inhalation of Dust

The time-averaged COI concentration in air, rather than COI intake, is used as the basis for estimating absorbed dose based upon guidance described in EPA [EPA, 2009].

$$C\_a = \frac{C\_s \times BF\_inh \times ET \times EF \times ED}{PEF \times AT}$$

where,

C_a = COI concentration in air, (mg/m^3)

C_s = COI concentration in soil (mg/kg soil)

BF_ing = bioavailability fraction from inhalation of dust (dimensionless)

ET = exposure time (hr/d)

EF = exposure frequency (d/yr)

ED = exposure duration (yr)

PEF = particulate emission factor (calculated; m^3/kg soil)

AT = averaging time (hr)

The PEF is in effect the volume of air occupied by one kilogram of respirable-size particulate matter (PM_{10}; 10 um and less), and is the output of dust resuspension and air dispersion models. If air samples are collected to directly measure concentrations in suspended dust, the PEF term is eliminated and the COI concentration term may be expressed as mg/m^3 of air.

The Hanover/Whitewater Creek IU HHRA ([Neptune, 2008]; Section 3.5.4) estimated PEF values for general wind erosion and vehicle-related erosion (e.g., for construction) of 1.34E+08 m^3/kg and 2.55E+06 m^3/kg, respectively. These values are used here for screening. Detailed derivation of these values can be found in the HHRA (Section 3.5.4).

Briefly, the general value assumes a size of 30 acres and a "grassland" ground cover. The value of 1.34E+08 m^3/kg corresponds to a concentration of respirable particulates in air of 0.0075 mg/m^3. The construction value again assumes a 30-acre area, but accounts for factors such as road area, vehicle characteristics, and so on. The value of 2.55 E+06 m^3/kg corresponds to a concentration of respirable particulates in air of 0.39 mg/m^3.

Appendix II: Descriptive Statistics for COPCs

COPC	Total Number	Nondetects				Detects				
		Number	Minimum	Mean	Maximum	Number	Minimum	Median	Mean	Maximum
Surface soil samples (0-1 inch) from LIU site										
Aluminum[1]	26	0	-	-	-	26	6870	13250	15451	29400
Arsenic[1]	26	0	-	-	-	26	1.4	3.4	5.1	28
Chromium[1]	26	0	-	-	-	26	5.5	12	22	99
Cobalt[2]	31	0	-	-	-	31	6.3	10	11	24
Manganese[1]	26	0	-	-	-	26	191	595	587	1440
Surface soil samples (0-1 inch) from LIU reference area										
Aluminum[3]	8	0	-	-	-	8	7370	10400	10693	15300
Arsenic[3]	8	0	-	-	-	8	1.0	4.4	4.2	7.2
Chromium[3]	8	0	-	-	-	8	6.3	11	11	16
Cobalt[3]	8	0	-	-	-	8	6.3	11	12	23
Manganese[3]	8	0	-	-	-	8	356	768	757	1430
Surface soil samples (0-1 inch) from STSIU/ERA reference areas										
Aluminum[4]	6	0	-	-	-	6	17800	22600	22567	28400
Arsenic[5]	9	1	1.4	1.4	1.4	8	0.7	2.4	2.3	3.1
Chromium[4]	6	0	-	-	-	6	24	34	36	48
Cobalt[4]	6	0	-	-	-	6	8.7	11	11	13
Manganese[4]	6	0	-	-	-	6	425	491	499	596

Sources:

1: Table 2-1 [Chino, 1995], 4 samples (excludes reference samples and duplicates); Table 2-16 [SRK, 2008], 1 sample; Table 4-1 [Arcadis, 2012], 21 samples.

2: Table 2-1 [Chino, 1995], 9 samples (excludes reference samples and duplicates); Table 2-16 [SRK, 2008], 1 sample; Table 4-1 [Arcadis, 2012], 21 samples.

3: Table 2-1 [Chino, 1995], 2 samples; Table 4-1 [Arcadis, 2012], 6 samples.

4: Table 2-1 [Chino, 1995], 6 samples.

5: Table 2-1 and Appendix C (page 328 of 386) [Gradient, 2008], 9 samples.

COPC	Total Number	Nondetects				Detects				
		Number	Minimum	Mean	Maximum	Number	Minimum	Median	Mean	Maximum
Shallow soil samples (0-6 inch) from LIU site										
Aluminum[1]	21	0	-	-	-	21	8320	14800	16793	29600
Arsenic[1]	21	0	-	-	-	21	1.1	3.5	5.5	36
Chromium[1]	21	0	-	-	-	21	4.4	17	21	63
Cobalt[1]	21	0	-	-	-	21	6.5	9.0	9.2	13
Manganese[1]	21	0	-	-	-	21	323	552	575	841
Shallow soil samples (0-6 inch) from LIU reference area										
Aluminum[1]	6	0	-	-	-	6	7260	10010	10548	14800
Arsenic[1]	6	0	-	-	-	6	0.7	5.1	4.3	8.5
Chromium[1]	6	0	-	-	-	6	4.3	15	13	18
Cobalt[1]	6	0	-	-	-	6	3.8	11	11	21
Manganese[1]	6	0	-	-	-	6	247	702	737	1650
Shallow soil samples (0-6 inch) from STSIU/ERA reference areas										
Aluminum[2]	6	0	-	-	-	6	32920	43610	46442	72320
Arsenic[2]	6	0	-	-	-	6	4.5	5.7	6.5	10
Chromium[2]	6	0	-	-	-	6	26	34	35	47
Cobalt[2]	6	0	-	-	-	6	7.0	15	14	20
Manganese[2]	6	0	-	-	-	6	911	1272	1291	1664

Sources:
1: Table 4-5 [Arcadis, 2012], 21 samples (excludes reference samples and duplicates).
2: Table 4-6 [SRK, 2008], 6 samples.

COPC	Total Number	Nondetects				Detects				
		Number	Minimum	Mean	Maximum	Number	Minimum	Median	Mean	Maximum
Sediment samples from LIU site										
Aluminum[1]	45	0	-		-	45	5110	8910	9809	19500
Arsenic[2]	53	9	0.9	18	20	44	0	3.8	4.2	19
Chromium[2]	53	0	-		-	53	4.2	9.2	19	86
Cobalt[3]	60	0	-		-	60	5.3	9.3	11	25
Manganese[4]	57	0	-		-	57	337	529	594	1420
Sediment samples from LIU reference area										
Aluminum	0	-	-	-	-	-	-	-	-	-
Arsenic	0	-	-	-	-	-	-	-	-	-
Chromium	0	-	-	-	-	-	-	-	-	-
Cobalt[5]	3	0	-		-	3	3.7	5.7	5.1	6.0
Manganese	0	-	-	-	-	-	-	-	-	-

Sources:

1: From [Arcadis, 2012]: Table 2-2 (excluded Tributary 2 locations 2204, 2205, 2208, 2209, 2210, 2212, 2213, 2228, 2229, 2231), 8 samples; Table 2-4, 4 samples (excluded Tributary 2 location ERA-34), Table 2-14, 24 samples, Table 2-17, 5 samples (from "metals" analysis), Table 4-8, 4 samples (excluded duplicate).

2: From [Arcadis, 2012]: Table 2-2 (excluded Tributary 2 locations 2204, 2205, 2208, 2209, 2210, 2212, 2213, 2228, 2229, 2231), 8 samples; Table 2-4, 4 samples (excluded Tributary 2 location ERA-34), Table 2-11, 8 samples; Table 2-14, 24 samples, Table 2-17, 5 samples (from "metals" analysis), Table 4-8, 4 samples (excluded duplicate).

3: From [Arcadis, 2012]: Table 2-2 (excluded Tributary 2 locations 2204, 2205, 2208, 2209, 2210, 2212, 2213, 2228, 2229, 2231), 16 samples; Table 2-4, 3 samples (excluded Tributary 2 location ERA-34), Table 2-11, 8 samples; Table 2-14, 24 samples, Table 2-17, 5 samples (from "metals" analysis), Table 4-8, 4 samples (excluded duplicate).

4: From [Arcadis, 2012]: Table 2-2 (excluded Tributary 2 locations 2204, 2205, 2208, 2209, 2210, 2212, 2213, 2228, 2229, 2231), 8 samples; Table 2-4, 4 samples (excluded Tributary 2 location ERA-34), Table 2-14, 36 samples, Table 2-17, 5 samples (from "metals" analysis), Table 4-8, 4 samples (excluded duplicate).

5: From [Arcadis, 2012]: Table 2-2 (excluded Tributary 2 locations 2204, 2205, 2208, 2209, 2210, 2212, 2213, 2228, 2229, 2231), 3 samples.

Appendix III: Acronyms

AOC	Administrative Order on Consent
BMD	Benchmark dose
COI	Constituent of interest
COPC	Constituent of potential concern
CSM	Conceptual site model
DP	Discharge Permit
EPC	Exposure point concentration
HEAST	Health Effects Assessment Summary Tables
HHRA	Human health risk assessment
HI	Hazard index
HQ	Hazard quotient
HWCIU	Hanover/Whitewater Creek Investigation Unit
ILCR	Incremental lifetime cancer risk
IRIS	Integrated Risk Information System
LIU	Lampbright Investigation Unit
LOAEL	Lowest observed adverse effect level
LSA	Lampbright Stockpile Area
MOA	Mode of action
NMED	New Mexico Environment Department
NOAEL	No observed adverse effect level
POD	Point of departure
PPRTV	Provisional peer-reviewed toxicity values
RfC	Reference concentration
RfD	Reference dose
RI	Remedial Investigation
RME	Reasonable maximum exposure
RSL	Regional Screening Level
SF	Slope factor
STSIU	Smelter Tailings/Soils Investigation Unit

SX/EW	Solution extraction/electrowinning
UCL	Upper confidence limit
UF	Uncertainty factor
UR	Unit risk
US	United States of America

Exposure Variables

AT	Averaging time
BF	Bioavailability fraction
BW	Body weight
DSA	Dermal surface area
DSAF	Dermal soil adherence factor
ED	Exposure duration
EF	Exposure frequency
ET	Exposure time
FS	Fraction site
IR	Ingestion rate
PEF	Particulate emission factor